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PHYTOPATHOLOGY

VOLUME 11 NUMBER 10

OCTOBER, 1921

THE MYCOPLASM THEORY—IS IT DISPENSABLE OR NOT?

JAKOB ERIKSSON

I. SORI AND MYCELIA OF RUST IN SEEDS

In the year 1911 Pritchard (Phytopath. **1**: 150–154.) and in the year 1913 Beauverie (Compt. Rend. Acad. Sci. Paris, 5 May, p. 1391, and 3 Nov., p. 787) published observations upon the occurrence of groups of rust spores and rust mycelia in American and in French seeds of wheat, barley and several cultivated or wild grasses (Bromus Agropyrum, etc.). The two observers presume that these sori might, after wintering, give origin to a new outbreak of rust in the following year. Two short replies by me against Pritchard in the year 1912 (Centralbl. f. Bakt. Abt. II, p. 453) and against Beauverie in the year 1914 (C. R. Acad. Sci. Paris, **158**: 1194) did not suffice for removing such an idea. In the years 1914 to 1918 Beauverie has continued publishing new articles in the same way (C. R. Acad. Sci. Paris, **158**: p. 1196, Trav. d. Biol. Veg., Paris, p. 11; Bull. Soc. Path. Veg. Fr. **5**: 89).

The most detailed and pretentious of Beauverie's articles is entitled "*Les germes des rouilles dans l'interium des semences des Graminees.*" In this he pronounces the following 4 theses: (1) the existence of mycoplasm is not proved; (2) sori of rust are to be found in the seeds of grasses; (3) these sori are sufficiently numerous; and (4) do these sori transmit the disease from one year to the other?

By the treatment of the three last theses he describes: (1) case of wheat, (2) cases of common barley and of *Puccinia glumarum* and (3) cases of *Bromus mollis* and of Agropyrum. For each of these cases he states the localization of the sori and gives numerous diagrams of seeds that are cut through. Neither in the text nor in the figures can I detect anything essentially new, that has not been mentioned in my book "*Die Getreideroste*" (Stockholm 1896). Also there are no proofs given for the transmission of the rust by means of such sori to the vegetation of the following year. This is also expressed in the following words by Beauverie (l. c., p. 12): "With regard to the thesis 4 ("Do these sori transmit the disease from one year to the other?") our researches and our experiences, though they have been numerous enough, do not permit us to pronounce today a definite answer to the question." In spite of that he can not leave the

hypothesis of transmitting the rust in that way and he discusses how the transmission may take place, either to the interior of the embryo or from the exterior on the young seedlings.

In order to prove the first alternative, i. e., the interior infection of the embryo, numerous experiments were arranged, but the results were "uncertain and insufficient." Beauverie considers the other alternative, i. e., the exterior infection of the young seedling, "much more likely" and he "hopes by means of a fall collection of seeds of barley, containing rust sori, to be able to solvė easily the problem the following spring."

The next publication by Beauverie upon this question is dated 1918. There he says, that the occurrence of the "intraseminal" sori varies considerably. They were very common in the years 1913, 1914, 1915 and 1916, but very rare in the years 1917 and 1918. This being the case, the propagation of the diseases by means of such seeds,—"if real?"—can only be "an adventive means" for assuring the perennity of the grain rust.

Thus it seems that the hope, pronounced by Beauverie in the year 1914 as to the definitive solution of the problem, was disappointing. For he does not report on any recent studies made by himself. He only refers to some experiments of Hungerford (Phytopath. 7: 73), performed at Madison (Wisconsin) in the year 1916. But these experiments gave negative results.

Such being the actual status of the question I think we can and must once for all, abandon the idea that the "intraseminal" sori in the seeds constitute a mode of hibernation of the grain rusts. The appearance of sori in grass seeds, is, as already stated by me in the year 1896, only a case of hypertrophic outbreak of the fungi without any importance whatever in their life-history.

II. THE STRUCTURE OF THE MYCOPLASM AND ITS TRANSITION TO MYCELIUM IN PHYTOPHTHORA INFESTANS AND PERONOSPORA SPINACIAE

While discussing the mycoplasm, the objection has been raised again and again, that I had not given a satisfactory analysis of the morphological structure of this mode of existence and of the transition of the intracellular mycoplasm to the intercellular mycelium. I hope my recent investigations upon the life-history of the late blight of potato (*Phytophthora infestans*)[1] and of the downy mildew of spinach (*Peronospora spinaciae*)[2] may satisfy all reasonable claims in that way.

[1] Eriksson, J. Uber den Ursprung des primären Ausbruches der Krautfäule, Phytophthora infestans (Mont.) de By. auf dem Kartoffelfelde. Ark. Bot., Stockholm, **14**: No. 20, 1916—Cfr. Rev. Gener. Bot., Paris, **29**: (1917) et **30**: (1918).

[2] Eriksson, J. Zur Entwickelungsgeschichte des Spinatschimmels (Peronospora Spinaciae (Grew) Laub.) Ark. Bot., Stockholm, **15**: No. 15, 1918.—Cfr. Rev. Gener de Bot., Paris, **32**: (1920). E. Foëx.

The research upon *P. infestans* was performed in the year 1915 with material of infected potato leaves, collected and imbedded in paraffin for cytological examination, in the years 1905 and 1911.

By the examination of the different zones of a spot on a diseased potato leaf, as seen at the earliest (*primary*) outbreak of the disease, in July or in August, I could follow all the evolution stages of the blight fungus from its life within the cells up until its penetration through the stomata as external mycelium developing aerial spores.

In the peripheric zone of the leaf spot I detected the fungus element, living with the protoplasm of the host cell. The two symbionts formed a colloidal fluid, including minimal granules and particles. The chlorophyll bodies become disorganized and assimilated in the surrounding plasm. The cell nucleus also is dissolved and in the place of it there appear several small nucleoles. Now the fungus element predominates in the symbiosis—this stage has changed to an *antibiosis*—and it is ripe and ready for issuing out from the cellar lumae into the intercellular cavities. It grows out as a plasm tube but this plasm tube soon changes its nature, consituting a mycelial thread. The mycelium develops organs for fructification (antheridia and oogonia). The results of this act become oospores. These are not resting spores, surviving to the next year for propagating the fungus. They are true summer spores, ready to germinate immediately and to produce mycelium on the surface of the plant.

For the research of the life-history of the down mildew of spinach I had at my disposition two different races of spinach, the one infected by the fungus and the other sound. The two races grew in the year 1911 at Experimentalfältet (Stockholm), the distance between the two races being only about 30 meters. On July 18, the first, i. e. the primary, spots of disease were observed on some leaves of the one race, and from that day new spots appeared successively on the same plant bed, while all leaves of the other race on the other plant bed continued to grow clean.

On the day of detecting the first spots, i. e., on July 18, small pieces of leaves from the diseased and from the sound plants were collected and imbedded in paraffin for cytological examination.

In the cells of the sound race I found a normal protoplasm with imbedded normal cell nuclei and chlorophyll bodies. In the peripheric zones of the spots of the diseased race, on the contrary, I detected that the cells were filled with a very roiled plasm between the chlorophyll bodies. These bodies as well as the nuclei became dissolved and assimilated in the plasm and small nucleoles appeared in the symbiotic plasm body. The whole evolution was exactly the same as that which occurred in the potato.

I was able to show, at a magnification of 2500 diameters, the different stages of evolution of the spinach mould in photomicrographic figures, which exclude every subjective construction. In some figures one can see the structure of the mycoplasm in the inactive stage in the infected cell, side by side with the structure of the normal protoplasm in a cell of the sound race. In other figures one can see a more advanced stage of the intracellular mycoplasm, its resting stage with nucleoles. In yet other figures one can see the discharge of the cell contents into the intercellular spaces and so on. The nature of the plasm tubes, just issued, may be seen very distinctly in several photomicrographic figures. If two tubes from two different cells meet one another they merge into one. The plasm tubes are a previous stage of an intercellular mycelium producing a secondary outbreak of the disease.

The diseased race of plants was after some weeks so roughly used by the fungus that the culture had to be pulled up and removed from the bed. The other race of plants grew sound until the ripening of seed. Seeds of this sound race, collected in the autumn of 1911, when grown in the year 1912 on several culture beds gave an exclusively sound harvest of spinach.

III. APPLICABILITY OF THE MYCOPLASM THEORY FOR OTHER PLANT DISEASES

Owing to my studies with cereal and hollyhock rusts and with potato and spinach blights, as well as on account of observations in the open air upon many other diseases since decades back, I am inclined to suspect the occurrence of a mycoplasm symbiosis in the life cycle of several other plant pathogenes. I mention a number of such: *Puccinia chrysanthemi, P. ribis, P. suaveolens, P. tragopogonis, Uromyces betae, U. alchemillae, Cronartium ribicolum, Phragmidium potentillae, P. subcorticium, Coleosporium campanulae, C. compositarum, Chrysomyxa abietis, Melampsora salicina, Peronospora trifoliorum, P. ficariae, Sphaerotheca mors-uvae, S. pannosa, Microsphaera euonymi, Plasmodiophora brassicae, tobacco mosaic, etc.*

STOCKHOLM

THE PATHOLOGY OF LUPINUS ARBOREUS, WITH SPECIAL REFERENCE TO THE DECAYS CAUSED BY TWO WOUND-PARASITES—COLLYBIA VELUTIPES AND PLEUROTUS OSTREATUS

ARTHUR S. RHOADS

WITH PLATES XVIII TO XX

INTRODUCTION

During the winter of 1918 the writer noted that the stems of *Lupinus arboreus* Sims, an arborescent shrub growing naturally on sand dune land in several widely separated localities in San Francisco, were frequently broken down as the result of decay. At various times during the rainy season he observed that *Collybia velutipes* (Curt.) Quél. and *Pleurotus ostreatus* (Jacq.) Quél. were of very common occurrence on stems of this species of lupine, both occurring as wound parasites and as saprophytes on dead stems (Plate 1). The common occurrence of these two wood-rotting fungi as active wound-parasites on this arborescent shrub, which has not been reported previously as a host for either of these fungi, presented to him a new and interesting problem that was deemed worthy of investigation.

CHARACTERISTICS AND ECONOMIC IMPORTANCE OF LUPINUS ARBOREUS

Lupinus arboreus Sims, commonly called the tree lupine or, by some, the yellow lupine, is an evergreen shrub becoming distinctly arborescent; it attains a height of from 4 to 8 feet and a diameter of as much as 4, or rarely 5, inches at the base. It is of common occurrence on sandy soils near the ocean, where, in the spring with its sulphur-yellow flowers in loose racemes often a foot in length, it is very showy.

Lupinus arboreus exhibits a great variety of growth forms. Occasionally it has an erect main stem, but more often it consists of several distinct stems which may be erect or nearly so, spreading well apart, or becoming more or less decumbent and sending up erect shoots from these. In its occurrence on an area it may vary from isolated bushes to dense thickets. Economically, *Lupinus arboreus* is of considerable importance as a soil retainer in sand dune country.

GENERAL PATHOLOGY OF LUPINUS ARBOREUS

Lupinus arboreus is a very rapid growing, comparatively short-lived shrub, readily attacked by a number of insects, parasitic fungi, and even animals.

Among the insects attacking *Lupinus arboreus* the most important is the larva of a moth, *Hepialus sequoiolus* Behr.,* whose tunnels the writer has frequently found in the living stem. Williams (12) mentions finding the larva most frequently in the large yellow lupine. He states that the larva bores longitudinal passages just above the ground or a little under, turning its burrow at right angles and usually closing the opening with excrement. He likewise states that *Hepialus sequoiolus* is not rare in the vicinity of San Francisco. A little later Williams (13) mentions finding the larvae in November feeding on the decumbent stems and in the thicker roots of the yellow lupine. In a third article Williams (14) contributes his observations on the life history of *Hepialus sequoiolus*, being chiefly an account of the pupation of the larvae. Williams states that they inhabit only old plants, boring into their thicker portions—sometimes going well into the roots and again ascending into the branches. Usually, however, they are found in the main trunk above or below the ground, a large decumbent stem being a particularly favorable locality for them. He likewise mentions finding in the same lupine stem the larvae of the large noctuid, *Gortyna immanis*, which is quite sluggish compared to *Hepialus sequoiolus*.

The tunnels commonly formed in the living lupine stems by these insects naturally afford excellent points for the germination of fungous spores and for the rapid spread of the mycelium through the stems. It is the writer's opinion that they are by far the greatest contributing cause to the destruction of *Lupinus arboreus* bushes by the two wood-rotting fungi discussed in the present paper.

Concerning the fungi occurring on *Lupinus arboreus*, a number of miscellaneous species of minor importance have been reported by various writers. Of the list enumerated below, all but one belong to the Ascomycetes and Fungi Imperfecti.

Cooke and Harkness (2, p. 16) list *Diplodia lupini* on stems. Phillips and Harkness (9, p. 25) describe *Stictus lupini* as a new species. Harkness (4, p. 44) describes *Diaporthe* (*Tetrastaga*) *lupini* on branches. Harkness (5) lists both *Dialonectria depallens* (p. 173) and *Sphaeria* (*Didymella*) *lupini* (p. 174) on stems. Harkness (6) lists *Stictus decipiens* Karst. on dead stems (p. 264), *Metasphaeria lathyri* Sacc. (p. 267), and *Pleospora leguminum* Rab. on legumes (p. 268); he also lists the following fungi from the Harkness herbarium: *Phoma lupini* (p. 268),

* Determination by Mr. G. F. Ferris of the Department of Entomology and Bionomics of Leland Stanford Junior University.

Sphaeropsis lupini (p. 268) *Hymenula lupini* (p. 270), and *Valsa lupini* (p. 270), all occurring on stems. Harkness (7, p. 445) lists *Sordaria languinosa* Sacc. on dead branches. With the exception of *Sphaeria (Didymella) lupini*, which was reported from Sacramento, the remainder of the above fungi were reported from San Francisco.

Farlow and Seymour (3, p. 27) record only the above mentioned species on *Lupinus arboreus*, but include *Macrophoma lupini* Berl. & Vogl. as a synonym of *Phoma lupini* Cke. & Hark., and *Nectria depallens* Berl. & Vogl. as a synonym of *Dialonectria depallens* Cke. & Hark.

To the foregoing list may be added the wood-rotting fungi, *Collybia velutipes* and *Pleurotus ostreatus*, which are of very common occurrence about San Francisco, and whose destructive action on the host is made the basis of this paper. The writer has also found a single sporophore of *Armillaria mellea* growing from the root of a living bush of *Lupinus arboreus*.

An orange-colored canker, caused by an undetermined species of imperfect fungus, is also of frequent occurrence as a parasite on the living twigs. This fungus gains entrance to the twigs through insect tunnels and develops an orange-colored, waxy stroma underneath the bark which, in time, splits longitudinally and recurves from the stem, often exposing more or less of the wood for a length of several centimeters. The hyaline spores, which are mostly 1-septate and falcate at maturity, are developed directly from the orange-colored, waxy stroma.

Among the animal injuries the depredations by mice are of most common occurrence. Their attacks are confined mainly to the smaller stems ranging from ½ to 1½ inches in diameter and usually are made at or near the base of the shrubs.

Even the very form of growth of the lupine bushes is often responsible for considerable mechanical injury. Crotches of the branches frequently split and, in large bushes where the stem branches close to the base, the luxuriance of growth, aided by the ease with which the stems split radially, frequently results in a more or less serious splitting of the stem. The splitting of the crotches and branches is materially augmented by people tramping around the lupine-covered dunes. These split crotches present ideal infection points for the spores of wood-rotting fungi and both *Collybia velutipes* and *Pleurotus ostreatus* were frequently observed fruiting at these points.

OCCURRENCE OF WOOD-ROTTING FUNGI ON LUPINUS ARBOREUS.

On January 1, 1920, the writer made an intensive reconnaissance of a portion of the lupine-covered sand dunes on the Fort Scott Reservation in San Francisco, in order to secure some idea of the percentage of shrubs

infected by these wood-rotting fungi. It is believed that the results obtained represent a fair average of the conditions existing at the time of the reconnaissance and that the figures will be sufficiently accurate to serve the purpose for which they were intended—namely to give an estimate of the percentage of shrubs bearing fruiting bodies of these fungi. The abundance of the sporophores, of course, may vary greatly from time to time, depending upon the weather conditions. The results of this reconnaissance are given in table 1.

TABLE 1

Number and per cent of Lupinus arboreus bushes on the Fort Scott Reservation in San Francisco bearing sporophores of wood-rotting fungi

Fungi	Number	Per cent
LIVING BUSHES		
Collybia velutipes	130	24
Pleurotus ostreatus	25	5
Armillaria mellea	1	0
Total bushes bearing sporophores	156	29
Total bushes not bearing sporophores	385	71
Total number of bushes examined	541	100
DEAD BUSHES		
Collybia velutipes	26	31
Pleurotus ostreatus	5	6
Total bushes bearing sporophores	31	37
Total bushes not bearing sporophores	53*	63
Total number of bushes examined	84	100

* In 23 of these bushes the stems were so badly rotted that they could be broken off by tramping against them. Doubtlessly a large majority of the remaining bushes were more or less rotted by at least one of these fungi.

In the above table, all bushes that had one or more living shoots from the root, were considered as living. The figures for the bushes bearing fungi do not necessarily indicate all of the infected stems, since, in the bushes not bearing sporophores, a number of stems were found to have rotten branch stubs and, when cut into, were found to be decayed within.

Subsequent observations indicated that by January 1 the fungi had attained their maximum fruiting. On January 4 it was noted that many of the sporophores were commencing to dry up, although a few fresh ones were beginning to make their appearance. On January 10, however, but few fresh sporophores of either *Collybia velutipes* or *Pleurotus ostreatus* were to be seen, practically all of them being well dessicated and more or less inconspicuous, especially in the case of *Collybia velutipes*.

1
2

In *Collybia velutipes* practically 90 per cent of the sporophores noted on the living bushes in table 1 occurred at the base of the stems, usually growing up from the root crowns (Plate XVIII, fig. 1). By excavating the sand about the root crown the development of the fruiting bodies could be seen readily. The sporophores, which usually occur in dense clusters, originate at some point on the root, often several inches beneath the surface of the ground. From this point, which may be an insect tunnel or the dead side of a root, there develop upwards a number of long attenuated, rhizomorph-like stems in all stages of growth. As a rule all stages in the development of the sporophore can be seen, varying from the slender stems bearing miniature unexpanded pilei that have not yet pushed up to the surface of the ground, to the stouter ones, which, when they reach the surface of the ground, abruptly expand into a stout velvety stem bearing the pileus. Occasional clusters are found where a number of rhizomorph-like stems have anastomosed into a single stem suggestive of the rooting base or pseudorhiza characteristic of *Collybia radicata*. The remaining 10 per cent of the sporophores noted on living bushes in table 1 developed on the stems at borer holes, branch stubs, split crotches, and dead areas at points varing from a few inches to 3 feet above the ground (Plate XVIII, fig. 1). The fruiting bodies that develop on the stems well above the ground lack the long attenuated rhizomorph-like stems and are considerably smaller than those that develop around the root crown where they receive the benefit of the soil moisture. In the dead bushes noted in table 1 all the observed sporophores of *Collybia velutipes* occurred at the bases, usually growing up from the roots underground.

In the case of *Pleurotus ostreatus* approximately 75 per cent of the sporophores noted on the living stems in table 1 occurred at points, such as insect tunnels, branch stubs, split crotches, and dead bark areas, from 1–3 feet above the ground. When developing on the stem, either at the ground line (Plate XVIII, fig. 2), or at points above it (Plate XIX, fig. 2), the sporophes invariably were laterally stipitate, with a poorly developed stem. Occasionally, however, the sporophores would develop from the roots beneath the surface of the ground; in this case they invariably were excentrically stipitate, with a well developed stem. As in *Collybia velutipes*, the sporophores developed up on the stems were much smaller and became desiccated more quickly than those developing at or near the ground line.

On February 21, 1920, after a comparatively long dry period, the writer again visited the lupine-covered sand dunes on the Fort Scott Reservation, to examine bushes of an associated species of lupine, namely, *Lupinus chamissonis* Esch., a blue-flowered species with silvery foliage, which occurs in small quantity there near the ocean. The stems of this

species are distinctly woody; however, they are much slower in growth and do not attain as large a size as those of *Lupinus arboreus*, none being noted that were over 2½ inches in diameter. The shrubs of *Lupinus chamissonis* are densely branched and very bushy, so that an examination of the stems for the presence of sporophores of fungi is quite difficult. However, the writer found three living bushes of this species with dessicated sporophores of *Collybia velutipes* that had developed at split crotches. This record of the occurrence of *Collybia velutipes* as a wound parasite on *Lupinus chamissonis* is also new.

A few days later the writer visited the sand dunes along Sloat Boulevard, near the ocean, where a large area is more or less densely covered with both *Lupinus arboreus* and *L. chamissonis*. Here one area was particularly striking in that practically all the bushes of *Lupinus arboreus* had been rotted by *Collybia velutipes* and *Pleurotus ostreatus*, whereas practically all the bushes of *Lupinus chamissonis* were growing vigorously and in a good healthy condition, only three being found bearing sporophores of *Collybia velutipes*. On an adjoining area, however, where there was practically a pure growth of *Lupinus chamissonis*, several bushes, both living and dead, were found bearing sporophores of *Collybia velutipes*. In so far as could be determined in the case of the living bushes, the sporophores always occurred in split crotches which are largely caused by human agency, there being no evidence of insect tunnels in the stems. Despite his persistent search, however, the writer has not found *Pleurotus ostreatus* fruiting on *Lupinus chamissonis*, although it very likely occurs on it.

In this connection it may be well to mention that, although the writer has collected the wood-inhabiting fungi rather extensively in the vicinity of San Francisco for more than a year, he has never found *Collybia velutipes* on any hosts but the two species of lupine previously mentioned. *Pleurotus ostreatus*, however, is encountered occasionally in the more humid forest sites. It is especially interesting, however, that these two fungi should occur so frequently as wound-parasites on the woody lupines.

On the sand dunes, both at Sloat Boulevard and on the Fort Scott Reservation, *Lupinus chamissonis* appears to be free from the insect injuries which are so prevalent in *L. arboreus* and which, in that species, lead to so many infections by *Collybia velutipes* and *Pleurotus ostreatus*. It also possesses a much tougher wood than *L. arboreus* and, by reason of this in conjunction with its more compact, bushy form, it is much more resistant to the natural mechanical splitting of the branch crotches which, in *L. arboreus*, is another great contributing cause to infection by the above-mentioned wood-rotting fungi. In case a permanent sand dune cover of lupine is desired, it is believed that *L. chamissonis* will prove of

far greater value than the larger, faster growing, but much shorter-lived species, *L. arboreus*, especially where the living shrubs are likely to become infected with such wood-rotting fungi.

REVIEW OF LITERATURE ON THE DECAYS CAUSED BY COLLYBIA VELUTIPES AND PLEUROTUS OSTREATUS

An extensive literature has been built up on both *Collybia velutipes* and *Pleurotus ostreatus*, particularly on the former species. It is the purpose of the writer to give only a brief resume of the more important literature on each species, in so far as it bears on the subject under discussion in this paper.

Collybia velutipes, commonly called the "velvety-stemmed Collybia," may be recognized readily by its yellow-brown pileus, which is viscid when moist; its stipe, which is dark brown and velvety at the base; and its habit of growing in more or less densely crowded clusters, both as a saprophyte on dead wood and as a wound-parasite on the roots and exposed wounds in living dicotyledonous trees, particularly species of *Ulmus*. It is unusual among the Agaricaceae in the early appearance of its sporophores, it being one of the few agarics that persist throughout the winter uninjured by freezing.

Biffen (1), who gives the most comprehensive account of the biology of this fungus, describes in detail the decay of the wood based on sections of culture blocks of horse-chestnut wood. According to him, *Collybia velutipes* attacks chiefly the cellulose portions of the wood elements, leaving a lignin skeleton. He concludes that soluble carbohydrates are of great importance in the proper nutriment of the fungus but that, in their absence, it is capable of varying its usual course of action and using lignin as a substitute. For example, when the wooden culture blocks, prior to their inoculation, were treated with caustic potash, which extracts the xylose-yielding substances, the sections cut from the wood, after its decay by the fungus, give a cellulose reaction with chloroiodide of zinc. In these sections Biffen found that the thickening layers stained a deep purple and were swollen so as to almost obliterate the lumen, the microchemical reaction being the same as given by wood infected by typical lignin-destroying fungi. He found that similarly extracted, but uninfected, wood gave no such reaction. He found, however, that if wood from which the xyloses had been removed be treated with a 1 per cent solution of cane sugar before infection, the action of the mycelium was similar to that in wood in which its chemical structure had not been artificially altered, that is, that the lignin was left unattacked.

Pleurotus ostreatus, commonly called the "oyster mushroom," usually occurs saprophytically but has been noted by various writers as a wound parasite on living trees, particularly shade trees. Its large, whitish to

dark gray, fruiting bodies are greatly esteemed by those who know their edible qualities—in fact, the lupine-covered sand dunes at Fort Scott, San Francisco, are frequently visited by one or more parties who are cognizant of the edibility of this fungus.

Learn (8), who gives the most comprehensive account of the biology of this species, describes in detail the decay of sugar maple (*Acer saccharum*) wood by this fungus. According to Learn, the results obtained for cellulose tests indicate that there is a gradual delignification of the cell-wall proceeding from the lumen outwards. As the cell-wall becomes delignified the lumen increases in size, the middle lamella being the last to be delignified in the advanced stages of the rot; in the very last stages of the decay even the middle lamellae may be broken down.

Learn found that the early wood of the annual ring became delignified much more rapidly than the late wood and that the medullary rays, which, in sugar maple wood, are several cells broad and strongly lignified, persist longer than the remaining elements. In addition he demonstrated the presence of diastase in the mycelium of the sporophore and made tests on ground rotten wood for hadromal, according to the methods employed by Czapek. From these he concluded that the hadromal is not entirely removed, while if there is a reduction of the hadromal it must be in very small quantities. The tests showed that the hadromal was still left behind in considerable quantities in the cell walls.

STRUCTURE OF THE NORMAL WOOD OF LUPINUS ARBOREUS

The wood of *Lupinus arboreus* is of extremely rapid growth, hard and close-grained, whitish, with heartwood absent, and possesses but little durability. When freshly cut it has a rather disagreeable leguminous odor.

Macroscopically the wood is of the ring-porous type, with exceptionally broad and rather indistinct growth rings, and inconspicuous pores. The pores, which lack tyloses, occur in more or less regular tangential lines and tend to become somewhat more numerous but not much larger in the early wood. On the ends of stems trimmed with a very sharp knife or razor the growth rings are demarked by what appears to be a more or less prominent tangential row of radially elongated vessels in the beginning of the annual ring. In seasoning this is accentuated by a slight honeycombing of the wood at this point. The medullary rays are very broad and conspicuous.

Microscopically there is no sharp line of demarcation between the different layers of growth. What appeared under the hand lens to be a row of radially elongated vessels proves to be the very thin-walled, hyalin medullary rays, which, particularly in the outer portion of the last growth

ring (in stems cut December 26), and to a lesser degree at the extreme periphery of each preceding one, consist of very thin-walled, hyaline cells entirely devoid of any layers of secondary thickening and of the numerous simple pits characteristic of the normally thickened ray cells. The growth rings are seen to be defined principally by the unlignified ray cells at their extreme periphery, and the gradual diminution of the vessels throughout the ring, both in size and numbers, which results in a slightly greater density of the late wood. Under the microscope the wood is seen to consist of the following elements: (a) multiseriate pith-rays, (b) comparatively small vessels, (c) wood fibers, (d) tracheids, and (e) wood-parenchyma fibers (Plate XX, A).

The multiseriate pith-rays are mostly 6–10 cells wide, high, conspicuous, and regularly disposed; the largest rays are as broad as the largest pores and their cells are homogeneous.

The comparatively small vessels occur singly or as more or less radial groups of from 2–4, disposed in irregular wavy lines. The individual vessels are composed of numerous relatively thin-walled, short tube-like segments arranged horizontally end to end, with more or less oblique end walls, particularly in the smaller ones. The sides in contact with other vessels are usually much flattened and bear numerous small bordered pits.

The wood fibers form the ground mass of the wood between the wide rays and occur in more or less compact masses alternating with more or less irregular, wavy lines of vessels. They can be distinguished readily from the other elements in transverse sections, microscopically, by their much thicker walls and, microchemically, by the lack of lignification in the laminae of thickening bordering on the lumen.

The tracheids are found adjacent to the vessels, together with the wood-parenchyma fibers. In transverse sections these elements can be distinguished readily from the wood parenchyma fibers by their much thinner walls.

MICROCHEMICAL TESTS OF SOUND WOOD OF LUPINUS ARBOREUS

As a preliminary to the detailed study of the decays by the two fungi here under consideration a series of microchemical tests with the most commonly used reagents was performed on sections of the sound wood of *Lupinus arboreus*, since it is believed that a knowledge of the relative cellulose and lignin content of the cell membranes, in conjunction with their anatomical features, will facilitate a clearer conception of the specific way in which the decay of the wood is brought about by the mycelium of these fungi.

The writer has refrained from giving in detail the specific reactions of the cell walls to the various reagents employed for the detection of lignin and cellulose, since they offer nothing fundamentally new.

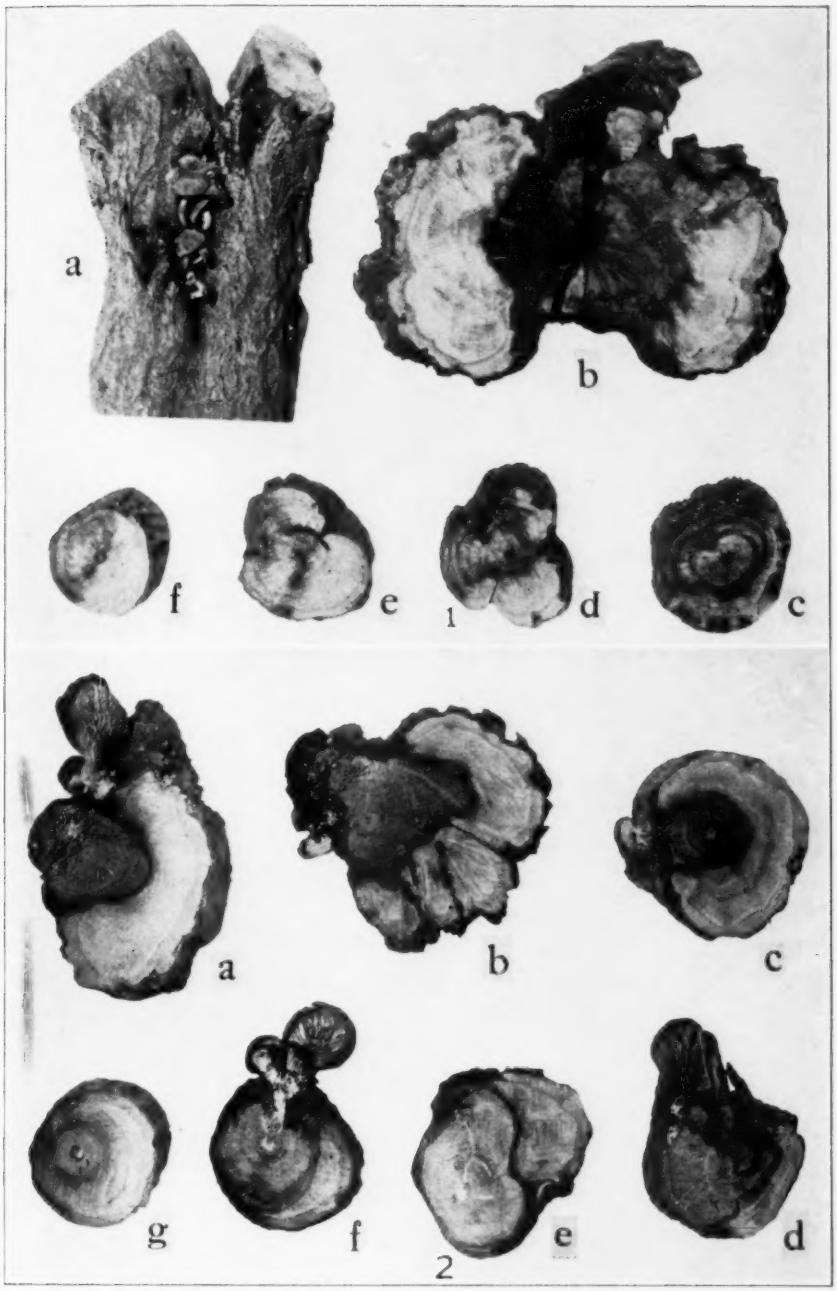
All the microchemical tests used agree with one another, not only in their differentiation of the cellulosic and lignified membranes, but also in the degree of lignification exhibited by the different wood elements. The results of these tests indicate that the unthickened middle lamella of the younger medullary ray cells and the laminae of thickening bordering on the lumina in all the wood fibers consist largely of cellulose and that the entire walls of the vessels, normally thickened medullary ray cells, tracheids, wood-parenchyma fibers, and middle lamellae and secondary laminae of thickening in the wood fibers are strongly lignified. Of the wood elements whose membranes are lignified, the vessels and medullary ray cells are by far the most strongly lignified, the vessels appearing a trifle more so than the ray cells. The remaining elements, namely the tracheids, wood-parenchyma fibers, and middle lamellae and secondary laminae of thickening in the wood fibers appear to be lignified to approximately the same extent.

THE DECAY OF LUPINUS ARBOREUS STEMS BY COLLYBIA VELUTIPES AND PLEUROTUS OSTREATUS

The course of the decay of *Lupinus arboreus* stems by *Collybia velutipes* and that by *Pleurotus ostreatus* were each worked out in full detail, using large numbers of microtome sections of the wood in all stages of the decay. The decays of lupine wood by these respective fungi were found to be practically identical in their character, differing only in the late stages of the rot in that the decay caused by *Pleurotus ostreatus* progresses somewhat farther than that caused by *Collybia velutipes*. For the sake of brevity, therefore, but the one description will be given in which the few points of difference between the two decays will be pointed out specifically.

The first evidence of decay in the living stem is the occurrence of a slight brown discoloration, usually irregular in outline and very striking against the background of white sapwood. This brown discoloration invariably starts at some dead branch stub, insect tunnel, split crotch or other mechanical injury, and from this point spreads up and down the stem as it gradually develops into the typical rot.

The decay spreads up and down the stems much faster than it does across them. It may start somewhere near the center of the stem and work outward until the periphery is reached, or it may start on one side of the stem (Plate XIX, figs. 1 and 2), killing this side as it extends up and down, and gradually extending across the stem until the whole of it is decayed. In the latter case one or more growth rings may be formed partially around the stem before the whole of it, as seen in cross section, becomes rotted by the advancing fungus.


a
b
f
e
1
d
c
a
b
c
g
f
2
e
d

When infection, by either of these fungi, occurs in a lateral branch the decay invariably is transmitted to the main stem. When infection occurs near the base of the stem the rot invariably is ultimately transmitted to the other stems of the bush, in case it has more than one stem.

Sections taken through the brown discolored wood, which, as stated previously, represents the first stage of the decay, show no perceptible change in either the physical or chemical structure of the wood itself. A microscopic examination of such sections shows the presence of mycelium occurring more or less abundantly in the vessels and some of the adjoining elements, but not of general occurrence throughout the wood; also that the enzymatic activity of the fungus is evident since the wood elements containing the hyphae are usually full of a brownish black decomposition product. This substance, as stated by the writer (10 and 11), is regarded as a humic by-product of the decay in the wood and usually starts as a result of the transformation of the cell contents by the action of the mycelium before the dissolution of the walls of the wood elements has begun. Evidence of the latter is offered by the result of microchemical tests made on sections of the brown discolored wood. The standard microchemical tests employed for the recognition of cellulosic membranes, as well as those commonly employed for the recognition of lignified membranes, all agreed in demonstrating that the wood in this incipient stage of the decay behaves exactly as does the normal wood.

Following along the longitudinal section of the diseased stem, often but a very short distance, the brown discolored wood is seen to gradually merge into the typical, lighter brown rot. Sections of wood cut through this transition point in the decay show that the brown decomposition products usually tend to become so abundant as to form a zone of varying width and regularity between the decayed and the undecayed wood. Under the microscope it is seen that the brownish black decomposition products become so abundant as to more or less completely fill the lumina of all the wood elements. This zone of brownish black decomposition products which delimits the decayed from the undecayed wood always keeps pace with the progress of the decay. These by-products of the decomposition apparently are more or less completely destroyed by the mycelium while new ones are formed immediately in advance of the hyphae invading the living wood.

Just behind the advancing zone of brownish black decomposition products, which roughly marks the limits of the advancing mycelium, can be seen the first stage in the dissolution of the cell walls. The first evidence of this occurs in certain of the wood fibers where it is noted that the tertiary laminae of thickening (the cellulosic laminae bordering on the cell lumen) in these elements gradually diminish in thickness until, but a very short distance back of the dark brown zone bounding the de-

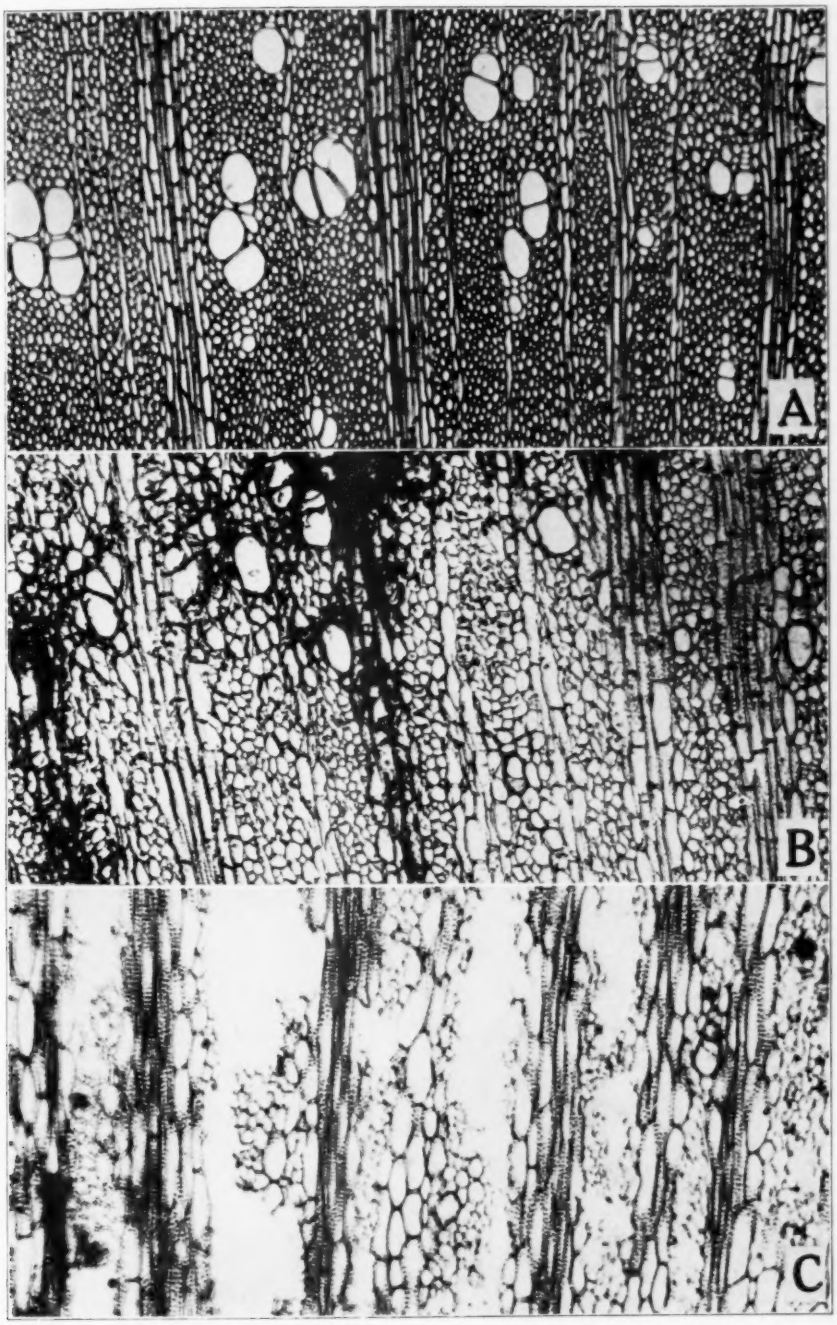
cayed area, this layer is seen to be dissolved completely in practically all the wood fibers. A repetition of the microchemical tests previously made on sound wood for the detection of lignin showed that the middle lamellae and secondary laminae of thickening in the wood fibers, together with the entire walls of the other elements, still respond as strongly as they did in the tests made on sound wood. Microchemical tests for cellulose, however, gave positive results only where the cellulosic layers, bordering the lumina of the wood fibers, still remained intact.

As the decay progresses the rot column enlarges, both longitudinally and in transverse area. As a rule, however, it is always characterized by the dark brown zone of decomposition products between its periphery and the adjoining sound wood.

Proceeding still farther back from the margin of the decayed wood, it is evident that the secondary layers of thickening in the wood fibers, tracheids, and wood parenchyma fibers are becoming dissolved progressively until, but a short distance farther, only the middle lamellae of these elements remain (Plate XX, A, B). At this point in the decay of the wood many of the wood elements between the medullary rays, with the exception of the vessels, have a skeletonized appearance. The middle lamellae of the dissolved cell walls retain the nature of lignified membranes and prove quite resistant to decay, but ultimately become so thin that, in parts of the sections, they were found to be broken up into dissociated fragments, probably under the stress of sectioning. At this stage of the decay the vessels and ray cells still respond to microchemical tests for lignin almost as strongly as they did in the sound wood. These elements, which originally were strongly lignified, appear to remain almost intact until the intervening wood elements are practically entirely skeletonized.

By the time the decay has progressed to this stage the rot usually extends completely out to the bark in case it started at the center of the stem, or, in case it started at one side of the stem, the majority of the living wood, as shown in cross section, has become well decayed. Stems rotted at or below the ground level usually can be broken off with comparatively little effort, even before the decay has progressed this far, providing that the decay has extended clear across the stem. With the completion of the decay in the stem the black zone, formerly appearing in advance of the rot column, is usually found to have disappeared from the stem.

Lupinus arboreus wood decayed by *Collybia velutipes* rarely becomes soft and pith-like as does that decayed by *Pleurotus ostreatus*, and then only in small localized areas where the decay, for some reason or other, progresses beyond the average for the whole stem. Whole stems never become soft and pith-like and white as do those decayed by *Pleurotus*


A
B
C

ostreatus. In the decay caused by *Collybia velutipes* the delignification of the wood elements usually does not progress to the same extent as in that caused by *Pleurotus ostreatus* and the decay seems to have run its course long before the wood substance is thoroughly destroyed.

In the decay caused by *Collybia velutipes* the wood elements, as a whole, do not become dissolved down to their middle lamellae, as readily, or at least not as quickly, as in the rot caused by *Pleurotus ostreatus*. In the former species the decay usually develops entirely across the stem before the wood elements are seriously broken down. Dead stems bearing clusters of sporophores at their bases, as a rule, can be broken off transversely with comparatively little physical effort. However, the yellowish-brown decayed wood is still found to be fairly hard. A microscopic examination of the wood at this stage of the decay shows that the majority of the wood elements are rarely dissolved down to their middle lamellae but still retain much of their secondary laminae. It also frequently happens that a portion of the brown by-products of decomposition is left behind in the decayed wood, particularly in the lumina of the vessels and adjoining elements (Plate XX, B). They are so scattered, however, as to be evident only when the section is examined under the microscope.

In decayed roots dug out of the sandy soil, where the maximum amount of decay is to be expected, the late stages of decay are to be found. At this stage the decayed wood is soft and spongy and brownish white in color. The annual rings of growth often separate readily and the wood can be broken readily in all directions, between the fingers. When dry it is light in weight and very punky; it can then be crumbled between the fingers to a fine powder.

By the time the wood has reached the advanced stage of decay just described, it invariably is discolored. It is reasonably certain that in lupine wood decayed by *Collybia velutipes*, continued destruction beyond this point is largely the work of other agencies. After a stem becomes rotted clear through and its death ensues, it is but a short time until the partially decayed wood is tunnelled into by ants and other insects.

In the decay caused by *Pleurotus ostreatus*, as soon as the decay progresses beyond the initial dark brown discoloration, the wood becomes yellowish brown in color. In the extreme stages of the decay the wood tends to become white and pith-like. The pith sometimes is replaced by a solid core of white mycelium and small white mats of mycelium frequently develop in the clefts formed in the decayed wood (Plate XIX, fig. 2, e), an occurrence never noted in the rot caused by *Collybia velutipes*. As in lupine wood decayed by *Collybia velutipes*, however, brown by-products of the decomposition are occasionally left behind in the decayed wood, particularly in the lumina of the vessels and the adjoining elements, although they are not to be found in any appreciable quantity.

When these late stages of the decay are reached the dissolution of the ray cells and vessels begins (Plate XX, C). At this stage of the decay the fine, colorless mycelium is seen to have spread throughout the wood substance, the individual hyphae penetrating the cells in all directions. By this time the wood is whitish and pith-like, tending to break readily both transversely and longitudinally. When wet it can be compressed at right angles to the pith rays by slight pressure between the fingers, since all the wood elements between the rays have become skeletonized, and the water quickly runs out of it; as soon as the pressure is released the water is instantly absorbed, as in a sponge. The decayed wood is most resistant to radial compression, under which it tends to break up into a series of radial sheets—the medullary rays—which resist decomposition longer than the wood fibers which form the ground work of the wood. After the pith-like decayed wood has become dry it becomes much more resistant to radial compression, under which the rays crumple, much as a deck of cards will do when compressed with sufficient strength. When dry, wood in this stage of the decay can be reduced to a flaky powder by rubbing between the fingers.

From the above study it is evident that the first main chemical change brought about by the action of the mycelium, in the case of both fungi under discussion, is the dissolution of the cellulosic layer bordering on the lumina of the wood fibers. The dissolution of this layer starts at the lumen and progresses toward the middle lamella. After the available cellulose has been digested the dissolution of the lignified membranes remaining occurs in the same way, beginning first with the ones least lignified. In this dissolution of the lignified membranes no free cellulose could be detected at any time during the course of either decay, although in the decay of *Acer saccharum* wood by *Pleurotus ostreatus*, Learn (8) states that "the reduction of the lignified tissue to cellulose is a very slow process."

The course of the destruction of *Lupinus arboreus* wood by *Collybia velutipes* agrees essentially, in so far as the salient features are concerned, with that described by Biffen (1) for horsechestnut wood; the course of the destruction of *Lupinus arboreus* wood by *Pleurotus ostreatus* likewise agrees essentially with that described by Learn (8) for *Acer saccharum* wood.

SUMMARY

Collybia velutipes and *Pleurotus ostreatus* are reported as serious wound-parasites on *Lupinus arboreus*, causing widespread destruction of this arborescent shrub which grows naturally on sand dune land in the vicinity of San Francisco, where it is of considerable economic importance as a soil retainer. In one case *Armillaria mellea* was also found attacking *Lupinus arboreus*. Occasionally *Collybia velutipes* also is a wound parasite on *Lupinus chamissonis*. These two species of lupine are here reported for the first time as hosts for these fungi.

Infection of *Lupinus arboreus* stems by these fungi is largely due to tunnels made in the stems by the larvae of the moth, *Hepialus sequoiolus*, and to such mechanical injuries as split crotches, which are of frequent occurrence, and to dead branch stubs.

As the result of a reconnaissance of a portion of one area of dune land covered by *Lupinus arboreus* growth it was found that, out of a total of 541 living bushes, 156, or 29 per cent, exhibited sporophores of wood-rotting fungi, as follows: 130, or 24 per cent, with *Collybia velutipes*; 25, or 5 per cent, with *Pleurotus ostreatus*, and 1 with *Armillaria mellea*. On this same sample plot 84 dead bushes were found. Of these, 26, or 31 per cent, exhibited sporophores of *Collybia velutipes* and 5, or 6 per cent, exhibited sporophores of *Pleurotus ostreatus*. Most of the remaining 53 dead bushes exhibited more or less decay, many of them being completely rotted. The production of the sporophores of these fungi was in direct relation to the amount of precipitation.

The decays of *Lupinus arboreus* wood by *Collybia velutipes* and *Pleurotus ostreatus* were found to be practically identical in character, differing only in the late stages of the rot in that the decay produced by *Pleurotus ostreatus* progresses somewhat farther and causes the wood to become whiter than that produced by *Collybia velutipes*. The first main chemical change brought about by the action of the mycelium in the case of both fungi is the dissolution of the cellulosic layers bordering on the lumina of the wood fibers. The dissolution of this layer starts at the lumen and progresses toward the middle lamella. After the available cellulose has been digested the dissolution of the lignified membranes remaining occurs in the same way, beginning with the ones least lignified.

By reason of its greater longevity, its tougher wood and more compact form, both of which qualities retard the splitting of the branch crotches, and its freedom from attacks by boring insects, *Lupinus chamissoni*, will be found decidedly preferable to the associated species, *L. arboreuss* for a permanent sand dune cover.

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PLATE XVIII

Fig. 1. Occurrence of *Collybia velutipes* on living shrub of *Lupinus arboreus*, showing a cluster of sporophores that have developed from the junction point of two stems below the ground line and a cluster that have developed out of a wound on the stem at the right.

Fig. 2. Occurrence of *Pleurotus ostreatus* on a dead shrub of *Lupinus arboreus*, showing abundant young sporophores developed from the common base of the stems.

PLATE XIX

Fig. 1. Sections of living stems of *Lupinus arboreus* attacked by *Collybia velutipes*: *a*, sporophores developing out of split crotch; *b*, section just below another split crotch,

showing sporophore and rot; *c*, *d*, *e*, and *f*, sections showing rot in living stem at ground line and at points one, two, and three inches above the ground-line. × ½ natural size.

Fig. 2. Sections of living stems of *Lupinus arboreus* attacked by *Pleurotus ostreatus*: *a*, section just below forks with dead rotten branch, showing sporophore and progress of rot down main stem, resulting in the early death of one side; *b*, section of same stem at point three inches below *a*, showing small sporophore at one side of dead wood; *c*, section of same stem 11 inches below *b*, showing small sporophore at one side of dead wood; *d*, section through another infected stem at point of emergence of dead branch stub, showing sporophore developed out between bark and wood; *e*, section through still another infected stem, taken just below fork in stem, and showing the complete decay of branch on left side with pith replaced by a core of mycelium, and spread of decay into the branch on right side; *f*, section of same stem at point one inch below *e*, showing sporophores growing out of rotten stub; *g*, section of same stem at point four inches below *f*, showing initial stages of the decay. × ½ natural size.

PLATE XX

A. Transverse section of normal wood of *Lupinus arboreus*, showing the character of the wood elements. × 68.

B. Transverse section of *Lupinus arboreus* wood decayed by *Collybia velutipes*, showing the dissolution of the secondary layers of thickening in the wood elements, and the dark brown decomposition products left in the wood. × 68.

C. Transverse section of *Lupinus arboreus* wood decayed by *Pleurotus ostreatus*, showing the dissolution of the secondary layers of thickening in the wood elements. × 68.

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RESISTANCE OF BARLEY TO HELMINTHOSPORIUM SATIVUM P. K. B.

H. K. Hayes and E. C. Stakman[1]

The spot blotch disease of barley has been known in the United States for about ten years. In 1910 Pammel, King, and Bakke described a new spot disease of barley caused by *Helminthosporicm sativum*. n. sp. Attention was called to the seriousness of the disease, and preliminary observations were made on varietal resistance. Later Johnson did considerable work on the disease and suggested (1913), as the common name, American blotch disease. Recently it has been called the spot blotch disease of barley.

It has been known for several years that the spot blotch disease is quite important in Minnesota. Since repeated infections may occur during the growing season, seed treatment will not eliminate the disease. It has been known for some time that the causal organism can attack wheat as well as barley. Recent work (Stakman, 1920) seems to indicate that *H. sativum*, or a form practically indistinguishable from it, commonly causes foot rot of wheat and rye in Minnesota and is capable also of infecting several wild grasses. Obviously, therefore, crop rotation and sanitation will not control the disease entirely.

Manchuria barley, which is the predominant variety in the Upper Mississippi Valley, is fairly resistant to the spot blotch disease. However, the long, rough awns make the barley crop very unpopular with the farmer. Hooded barleys are much easier to handle but do not yield well in humid regions. Furthermore, Harlan and Anthony (1920) have shown that the barley awn is an important physiological organ. The importance of smooth-awned barley was therefore recognized several years ago by Harlan, of the Office of Cereal Investigations, who recently (1920) described some importations of *Hordeum vulgare nigrum leiorrhynchum*, smooth-awned barleys. Lion, one of these smooth-awned barleys, was crossed with Manchuria with the hope of producing high-yielding, smooth-awned barleys suitable for Minnesota conditions. Harlan and Hayes (1919), in co-operative experiments between the Department of Agriculture of the University of Minnesota and the Office of Cereal Investigations of the United States Department of Agriculture, made an intensive study of several smooth-awned strains from crosses between Manchuria and Lion in comparison with high-yielding, rough-awned varieties. Three of these smooth-awned selections yielded well in the

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rod row nurseries at the Minnesota Experiment Station and at the substations, from 1916 to 1918 inclusive. In subsequent years, however, they have not yielded well. Very evidently this was largely due to the fact that they are susceptible to *H. sativum*. It was quite clear from casual observations that resistance or susceptibility to the spot blotch disease was an important factor in causing differences in yield between various strains and crosses of barley. For this reason a more careful study of varietal resistance has been made. Studies also are being made on the resistance of crosses between the rough-awned barleys and the smooth awned types.

Although the results presented are preliminary, the facts show quite clearly that barley varieties differ greatly with respect to susceptibility to *H. sativum*. It also has been shown that resistance and susceptibility are inherited, although the actual number and nature of inherited factors is not known.

TABLE 1

Correlation in barley between yield in bushels obtained from replicated rod row test and reaction to H. sativum. University Farm, 1920

Yield in bushels per acre	Reaction classes to H. sativum								
	L	L+	M−	M	M+	H−	H	H+	
15								1	1
27	1								1
30	1						1	2	4
33			1		1	1			3
36			1	1	2		1		5
39	1	1		1	1				4
42	3	2		1			4		10
45		4	1	1					6
48	2			2					4
51		2							2
54				1		1			2
57	2								2
66					1				1
	10	9	3	7	5	2	6	3	45

$r = -.36 \pm .09$

THE RELATION BETWEEN YIELD AND RESISTANCE

Varieties and crosses of barleys were studied both under natural conditions and after being subjected to an artificially induced epidemic of the spot blotch disease. A considerable number of commerical varieties and purified smooth-awned crosses were grown in five-foot rows in the Plant Pathology Nursery. An epidemic was induced in 1920 by spraying the plants repeatedly with a suspension of spores from pure cultures of *H. sativum*.

Data also were taken on the resistance of varieties grown in rod rows in the Plant Breeding Nursery in which only a natural epidemic developed. Three rows of each variety were grown in each of three different plots, systematically distributed over this nursery. The yields of the central row of each plot were determined and these were averaged for each variety.

TABLE 2

Correlation between manner of reaction of 45 barley varieties to H. sativum under artificially produced epidemic conditions in the plant pathology plots and the manner of reaction in the plant breeding nursery. 1920

Manner of reaction, plant breeding nursery	Manner of reaction, plant pathology plots: T+	L−	L	L+	M−	M	M+	H−	H	H+	
T+							1				1
L			4	1	3	2					10
L+	3	2	2	1	2						10
M−								1			1
M			3	1	2	1	2				9
M+				2		3					5
H−					1						1
H					1	2	1	1		1	6
H+					1				1	1	2
	3	2	9	5	10	8	4	2	1	2	45

$r = .49 \pm .08$

The severity of attack by *H. sativum* was denoted by the following words: trace, light, medium, and heavy. Plus and minus signs were used to indicate plus or minus variations within each respective class. No variety was entirely immune to the spot blotch disease, but there were sharp differences in the susceptibility of different varieties and crosses. All of the strains were selections or crosses which, on the basis of previous tests, seemed to be promising under Minnesota conditions.

A correlation coefficient was computed between the manner of reaction to *H. sativum* and the average yield of the varieties and crosses. (See table 1.) While only forty-five forms were used in the test, the use of the correlation table is justified, since it presents clearly and concisely the extent of the apparent relation between susceptibility or resistance to Helminthosporium and the yield in bushels per acre. The computed correlation coefficient was $-.36 \pm .09$. This shows that in general the resistant varieties yielded best. Yield may be considered the end result of the interaction of numerous inherited or environmental factors, among which disease resistance is one.

Some varieties and strains were grown both in the Plant Pathology Nursery and in the Plant Breeding Nursery. The reaction of these plants to *H. sativum* is shown in correlated form in table 2. Some of these varieties are the same as some of those for which which data are given in table 1.

The degree of infection of the different varieties in the two nurseries was approximately comparable. However, the correlation coefficient. $.49 \pm .08$ is not especially high. No sharp line of differentiation between the different classes of infection could be drawn. The various types of infection formed a closely intergrading series. Those varieties, however, which were slightly infected in one nursery were infected in a somewhat similar manner in the other nursery and vice versa. This indicates that the difference between classes is genetic.

TABLE 3

Correlation between average manner of reaction to H. sativum in 1920 and yield under replicated rod row test in 1919

Yield in bushels per acre, 1919	Reaction classes to H. sativum								
	L	L+	M−	M	M+	H−	H	H+	
18				1		1		1	3
21			1		1				2
24			1			1			2
27						2		1	3
30							1		1
33							1		1
36	1	1	1						3
39				1					1
	1	1	3	2	1	4	2	2	16

$r = -.38 \pm .14$

No data were taken on the degree of infection in the rod row plots in the Plant Breeding Nursery in 1919, although many varieties were heavily infected. Some varieties which were grown in the Plant Breeding Nursery in 1919 also were grown in 1920 and data were taken on their susceptibility to *H. sativum*. Sixteen varieties were grown both in the Plant Breeding Nursery and in the Plant Pathology Nursery in 1920. The reaction of these varieties to *H. sativum* in the two tests was averaged and this result was correlated with the results of the yield tests in the replicated rod rows in 1919. (See table 3.) It is evident from the table that the varieties which were resistant in 1919 in general yielded more than the susceptible varieties.

COMBINATION OF RESISTANCE TO H. SATIVUM AND THE SMOOTH-AWNED CHARACTER IN A SINGLE VARIETY.

Since no entirely satisfactory smooth-awned variety was obtained from the first series of purified crosses, more crosses were made between smooth-awned barley and various six-rowed forms. These crosses were made by Dr. H. V. Harlan of the Office of Cereal Investigations, United States Department of Agriculture, at Aberdeen, Idaho, in 1917. The parental forms were pure lines, and the F_1 crosses were grown in the greenhouse of the United States Department of Agriculture at Washington in the winter of 1917 and 1918. The subsequent study, from F_2 to F_4 inclusive, was made at the Minnesota Experiment Station. Fifty plants of several F_3 familes were grown in 1919. All of these familes were grown from smooth-awned plants selected in 1918, and they bred true for the smooth-awned habit. The awns of some of the plants, however, are smoother than those of other plants. All of the smooth-awned plants have a few sharp projections at the base of the awn and on the upper third. These projections, however, are much shorter than those on the tooth-awned varieties.

TABLE 4

Manner of reaction under artificial epidemic conditions of smooth and tooth awned barley parents and sixteen F_4 families to Helminthosporium sativum

Parents and F_4 Purified crosses	Reaction classes						
	L	L+	M—	M	M+	H—	H
Tooth-awned parents	3	4					
Smooth-awned parents						1	2
F_4 families	2	2	3	3	3	1	

There was a very clear difference in the resistance of the different F_3 families to *H. sativum*. The plants in some of the families were extremely susceptible. Those of others were resistant, and in some of the families some plants were resistant while others were susceptible. The plants of some of the F_3 families apparently were uniform for general agronomic and botanical characters and also were resistant to *H. sativum*. The smooth-awned, pure line parents of these crosses were highly susceptible in 1920 both in the Plant Pathology Nursery and in the Plant Breeding Nursery. The rough-awned parents, on the other hand, were highly resistant.

Seventeen F_4 families were grown in the pathology plots. Sixteen of these appeared to be homozygous in their manner of reaction to *H. sativum*. These families had been selected in the F_3 because they seemed to be desirable types. The reaction of these sixteen F_4 families and the

reaction of their parents are given in table 4. It is evident from these results that resistance and susceptibility to *H. sativum* segregate in barley crosses between resistant and susceptible parents. The results show also that, by crossing, a variety may be obtained combining the resistance of the rough-awned parents with the smooth-awned character of the smooth-awned parents.

RESISTANCE OF COMMERCIAL BARLEY VARIETIES

A preliminary study was made of the relative resistance of commercial varieties of barley. The results are given in table 5.

TABLE 5

Comparative resistance of commercial barley varieties to Helminthosporium sativum

SPECIES[1] AND VARIETY	COMMERCIAL VARIETY	DEGREE OF INFECTION
Hordeum vulgare pallidum	Manchuria	
	Pure line 1	M
	Pure line 2	M—
	Pure lines 3 and 4	L+
	Pure lines 5–10 inc.	L
Do	Servian	H
Do	Trebi	M+
Do	Odessa	H—
Do	Bay Brewing	H
Do	Tennessee Winter	L
Do	Winter Club	M—
Do	Mariout	H+
Hordeum vulgare nigrum	Gatami	L
Hordeum vulgare horsfordianum	Horsford Beardless	M+
Hordeum vulgare coeleste	Himalaya	M+
Do	Black Hull-less	H—
Do	Nepal x Manchuria (Several purified crosses)	L
Hordeum vulgare trifurcatum	Nepal	L
Hordeum distichon palmella	Hanna	L
Do	Chevalier	L
Do	Hannchen	L
Do	Svanhals	L

[1]The classification proposed by Harlan has been used.

SUMMARY

1. During the progress of breeding work, undertaken for the purpose of producing smooth-awned barleys, the importance of resistance to the spot blotch disease, caused by *Helminthosporium sativum*, became very apparent.

2. Crosses were made between Lion, a smooth-awned variety susceptible to *H. sativum*, and Manchuria, a rough-awned, resistant variety.

3.The first group of homozygous crosses which were increased and tested did not yield well. This apparently was due to the fact that these crosses were as susceptible to *H. sativum* as is Lion, the smooth-awned parent.

4. After the failure of the first group of crosses, further crosses were made between Manchuria and Lion. A considerable number of the resulting F_3 smooth-awned families were grown in 1919, and a fairly severe epidemic of the spot blotch disease developed. Some F_3 families were as resistant as the Manchuria parent; others were as susceptible as the Lion parent; and in some of the families both resistant and susceptible plants appeared.

5. Some F_3 families were selected in 1919 and were tested carefully in 1920. Some families were obtained which combined desirable botanical and agronomic characters, including smooth-awns, with resistance to *H. sativum*.

6. A preliminary study was made in 1920 of the resistance and susceptibility of commercial varieties of barley. From the results obtained it seems reasonable to conclude that resistant barleys of any desired botanical group of Hordeum can be produced.

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VIOLET ROOT ROT (RHIZOCTONIA CROCORUM DC.) IN THE UNITED STATES

JAMES A. FARIS[1]

INTRODUCTION

During the fall of 1919 specimens of potatoes covered with a violet felt of mycelium were received by the Department of Pathology of the University of Nebraska. These diseased tubers came from Sheridan County where the growers reported having observed the disease over a period of three years. Further investigations showed the disease present on potatoes in Kimball and Cherry Counties in the same state. The experimental plots at Gordon, Nebraska, were heavily infected, even though the seed tubers had been treated for black scurf (*Rhizoctonia solani* Kühn) with mercuric bichloride solution (1–1000).

During the growing season of 1920 the writer made numerous observations of plants infected with *Rhizoctonia crocorum* DC. Through this entire season the disease was very prevalent in the potato growing region of northwestern Nebraska.

PREVIOUS REPORTS OF VIOLET ROOT ROT IN THE UNITED STATES

The following table shows a steady increase in the known range of this fungus throughout the United States:

TABLE 1

Violet root rot in the United States

YEAR	HOST	AUTHOR	STATE
1915	Solanum tuberosum	Bailey (3)	Oregon
"	" "	Rees (16)	Washington
1916	" "	Heald (13)	"
1889	Medicago sativa	Pound (17)	Nebraska
1900	" "	Tyler (21)	Arizona
1906	" "	Heald (11)	Nebraska
1908	" "	Freeman (9)	Kansas
1913–1914	" "	Reed & Crabill (19)	Virginia
1914 to 1919	" "	Johnson & Haskell (14)	Iowa, Kansas & Nebraska
1915	" "	Arthur (14) Jackson (14)	Indiana
1916	" "	Diehl (5)	Iowa

[1] The author is indebted to the University of Nebraska for the use of their pathological laboratories and greenhouses and to the staff of the botanical department for suggestions and criticisms.

From a review of the fragmentary reports on *Rhizoctonia crocorum* DC., we find that it has received some attention as causing a disease of alfalfa, and in one case (19) experiments were planned looking toward a possible means of combating the malady. As a disease of potatoes, reports have been confined to the appearance on the tubers. No survey or cultural work has been undertaken to determine whether or not other plants are serving as hosts for this parasite in America.

VIOLET ROOT ROT IN EUROPE

While reports of the disease due to this organism are comparatively recent in American literature, the situation in Europe is quite different. There it has been known for almost two centuries as the cause of destructive diseases upon many economic crops, as well as upon numerous wild plants.

For the present study it will not be necessary to attempt even a brief review of the European literature. For an extended discussion of this, the work of Duggar (6) should be consulted. References will be made to some of the leading features of the European results when they have a bearing upon the particular problem in hand. In a summary of the European literature Duggar (6) lists this fungus as parasitic on forty-seven genera, belonging to twenty-one different families of plants, mostly dicotyledons. The first report of this disease came from France (7), in 1728, where it was destroying *Crocus sativus*. From that time the fungus has been noted as attacking such economic crops as asparagus, alfalfa, carrots, red clover, and various other plants in southern and western Europe.

THE FUNGUS

Many changes in the nomenclature of this fungus have been made as the knowledge of the form has progressed. The lack of a known fruiting stage is, no doubt, responsible for the confusion and difference of opinion regarding the taxonomic position of the organism. A summary of the synonymy has been collated by Duggar. He regards it as unfortunate that *R. crocorum* DC. has priority over the more descriptive name, *R. violacea*, given by the Tulasne brothers (20). The most recent suggestions concerning its name have been made in a paper by Eriksson (8), and are based upon what he believed to be the perfect stages. After having given a review of the distribution of this fungus throughout Europe, Eriksson discussed his experiments with the asparagus and alfalfa forms, which he considered to be separate species. After an extended treatment of the results of this test, which was carried on for one year, he concluded that the violet root rot of the carrot and that of the alfalfa were two entirely different things. This, if true,

throws an interesting light upon the whole violet root rot problem. The experiments carried on by Eriksson are not accepted by all workers as giving conclusive proof of the convictions expressed in his paper. Eriksson's suggestion that biologic forms of this Rhizoctonia exist, and his conclusion that separate species infect carrots and alfalfa, needs to be confirmed by some careful cross inoculation experiments. The field for cultural work to determine whether we have biologic forms of this fungus as Eriksson suggests, and the range of host plants for such forms, if they exist, is as yet unexplored. No doubt the difficulty of securing the fungus in pure culture is responsible for this.

At the close of his paper Eriksson states that he has confirmed, to his own satisfaction, the connection of *R. medicaginis* with *Leptosphaeria circinans* by germination of the Leptosphaeria spores. The germ tubes, which he figures, he considers to be identical with the mycelium of *Rhizoctonis medicaginis*. It is indeed unfortunate that Eriksson did not perform infection experiments with these viable spores and give us the final and necessary test of the connection of the two forms.

MORPHOLOGICAL CHARACTERS OF THE FUNGUS ON THE POTATO

Mycelium. The younger external hyphae are almost colorless. Very soon the cytoplasm develops a faint violet pigment. As the hyphae grow older the walls of the cells become a brownish violet. The color in the walls gradually deepens until the older hyphal strands are a decided violet to buff color. The cytoplasm is conspicuously granular when very young but becomes more vacuolate with age. The young hyphal tips measure 12 to 14 microns in diameter, while the older hyphae measure 7.2 to 10 microns. The long conductive vegetative cells vary in length from 55 to 173 microns. The cells covering the superficial sclerotia are, as a rule, shorter (30 to 50 microns) and thicker (12 to 14 microns) than the cells of the conductive hyphae. In diameter they compare very favorably with the cells of the young hyphal tips. Branching is at right angles. A constriction may appear in the young branch at first but this is not evident in the older strands.

The characters of the internal mycelium are much the same as those of the external mycelium. These strands are septate and branched, the direction of the branching here being determined by the cells of the hosts. Even here there is a distinct tendency of the hyphae to branch at right angles. The internal mycelium on the stem does not differ materially from the superficial external mycelium as to size. There is very little pigment produced after the mycelium enters the host. This feature is conspicuous when an older colored hypha on the surface branches to form an internal hypha under the epidermis. The line of

demarcation of the pigment is rather clear cut. The internal mycelium is intercellular among the loosely associated cortex cells. The young hyphal tips enter some of the host cells and the formation of the internal sclerotia follows. While the mature mycelia of *R. crocorum* and *R. solani* would not be confused, the differences in very young hyphae are not striking.

Sclerotia. Within the superficial mantle of mycelium on the surface of the stem there soon appear reddish-black sclerotial bodies. These are longer than they are wide, and in some cases two are formed together, end to end. They appear within eight weeks after the infected tubers are planted and are very numerous on the stem from the ground surface to the seed piece, thirty-three having been counted within a distance of four inches. Surface sclerotia are also numerous at the angles of the roots and on the young rootlets, but always in the mantle of mycelium.

The sclerotia vary from the size of a pinhead, evidently very young ones, to 5 mm. in length. At this stage of development they invariably are longer than they are wide and raised about 1 mm. from the surface of the host. When macerated and examined under the microscope they show cells with all varieties of color from transparent to violet.

The superficial sclerotia are not firmly attached to the host but are connected with all the surrounding mycelial strands. The cells of the hyphae directly joining the sclerotial mass are, as previously described, somewhat thicker and shorter than the cells of the conductive mycelium.

The sclerotial cells proper show the peculiarly branched and curiously lobed condition described by Duggar (6). Under the microscope these cells were seen to be somewhat loosely packed together, giving on the whole, the appearance of a rather spongy, soft mass. In the sclerotia examined, particles of sand were usually included within the young sclerotium. The sclerotium became harder upon drying. This type of sclerotium was not abundant upon the original infected tubers, nor were they conspicuous upon the young tubers on the plants. Rarely was a superficial sclerotium found forming beneath the rudimentary leaves of the tubers.

Infection cushions. On badly infected tubers large numbers of small specks the size of a pin head were to be seen with the unaided eye. Transverse sections of a portion of the potato rind showed these to be submerged sclerotial masses from the tops of which many hyphae extended over the surface of the potato and also joined with those from other submerged sclerotia. These infection cushions were very close together, in some cases adjoining or nearly so. The formation of these

infection cushions has been carefully followed both on young stems and young tubers. A young hyphal branch penetrated directly through the wall of a host cell and once inside began to branch profusely. Typical sclerotial cells were formed in the host cell until finally the cell was completely filled by the fungus. The formation of the infection cushions in the young tuber takes place in exactly the same way. Subsequent growth of the fungus compresses the host cells immediately adjoining the sclerotial cells. The host cells in such infected tissue are disorganized. This disorganization increases as the internal sclerotia develop, until finally several cells are broken down and the cavities filled with the fungus mass. The tissue of least resistance lies to the outside of the sclerotial mass, and this is the first to be broken down. However, the pressure of the fungus mass tends to bend the walls of the lower cells downward. Bailey (3) interprets this downward bending as indicating that the fungus has pushed its way in from the surface. The writer has quite a different conclusion insofar as the formation of the infection cushion is concerned. The infection cushions have their beginning in the cork cambium from hyphal strands which have forced their ways to this host tissue by pushing between the cells of the epidermis. Such young infection cushions form in the cork cambium, and, as the cells of the cambium divide a few layers of cork tissue form below the sclerotia. Completely submerged sclerotia were not uncommon even in mature tubers. There is an abundant branching of the hyphae from the top of some of the infection cushions. There appears to be very little spread of the fungus threads from the base of the infection cushions. There is a suberized layer of host cells which stains red with fuchsin. When submerged sclerotia are examined under low magnification this layer gives one the impression of branching from the base of the infection cushion. The young sclerotial cells of the fungus have a dense granular cytoplasm which stains a deep red with both Pianese stain and acid fuchsin.

THE DISEASE

The disease caused by *Rhizoctonia crocorum* DC. is known as violet root rot, red root rot, or violet Rhizoctonia root rot. As suggested by the common name of the malady the fungus usually confines its attack to the underground parts of the plant. However, in a warm, humid atmosphere in the greenhouse the fungus extended up the stems and to the leaves of some of the potato plants. The favorable moist condition in this case was induced by placing a bell jar over half grown plants. The fungus, which lives in the soil from year to year, progresses slowly, killing patches from one to three rods in diameter in the alfalfa fields.

It has been noted by some observers that the soil where the disease occurred was acid in reaction. While this may favor the rapid development of the fungus, experiments where some infected potatoes were grown in a well limed soil do not seem to indicate that liming would prohibit its growth. The results of these experiments are given in table 2, where potato plants in five pots out of seven developed the disease when the soil was limed.

TABLE 2

Tabulated results of potato infection

POT NO.	SUPPLEMENTARY CROP	DEGREE OF INFECTION		
		ON STEMS	ON TUBERS	ON ROOTS
1	Alfalfa	Light—sclerotia small	Light	Light
2	Sugar beets			
3	Radish	Light		
4	Carrots			
5	Asparagus			
6	Alfalfa	Light	Light	Light
7	Sugar Beets	Light	Light	Light
8	Asparagus	Heavy—many sclerotia	Moderate	Moderate
9	Carrot	Light		
10	Asparagus	Moderate	Moderate	Light
11	Alfalfa	Light—few sclerotia	Trace	Trace
12	Sugar beets	Light	Light	Light
13	Radish	Light	Light	Light
14	Carrots	Light	Light	Light
15	Radishes			

SYMPTOMS OF THE DISEASE

Symptoms on alfalfa. The symptoms of the violet root rot on alfalfa have been described by Heald (13), Freeman (9), and others. According to these authors, at first a single plant becomes of a pale yellow color and soon after withers and dies. From this infection center, the fungus spreads in all directions, thus producing dead areas roughly circular in outline. The progress of this fungus, in common with other soil fungi, is slow, varying from three to twenty feet in a single year. In such spots a majority of the plants are killed outright. Practically all attacked plants are reported (9, 13) as losing their tap roots and, if they survive at all, they are not vigorous. In the more heavily affected plants the cortex of the root easily slips away from the vascular cylinder. This stage is followed by a complete rotting of the root which crumbles to pieces when the soil is disturbed.

As the disease progresses in the spots of the alfalfa field we have an area with a dead center surrounded by an area of dying plants, and farther out an area where there is an evidence of a yellowing of the foliage with a paler appearance and sometimes marked chlorosis of the leaves. Considerable wilting is sometimes exhibited at the time of the second cutting and this may come without previously evident symptoms.

The disease is characterized as causing a very rapid death soon after the first symptoms become noticeable. Duggar (6) states that the symptoms on the parts above ground are not striking and were it not for the dead areas in the field it would not be an easy matter to distinguish slightly affected spots.

The appearance of the violet mycelium on the roots is very striking and typical of this disease. The roots are completely enveloped with a crust-like mat of violet mycelium with numerous infection cushions, or sclerotia. Reed and Crabill (19) observed the purple coating to extend downward as much as seven inches on the root, and suggest that it probably extends to a greater depth. Specimens in the herbarium of the University of Nebraska show the Rhizoctonia strands covering the alfalfa tap root fourteen inches below the crown.

Symptoms on the potato. Plants from infected tubers planted in the greenhouse February 29, 1920, in a soil free from the disease began to show yellowing and wilting of the lower leaves which arose from the stem below ground. These leaves were covered with the violet felt mycelium which showed all gradations in color, including transparency of the younger tips, pinkish brown, rose brown, and in the older hyphae a violet color. These leaves soon dropped. Outside of this dying of the lower leaves, the plants appeared perfectly healthy and produced a very vigorous top growth. The dying of these leaves could not with certainty be attributed to the attack of the Rhizoctonia.

Plants lifted from the soil nine weeks later, showed the entire underground part of the stem to be covered with the rose brown mycelium which assumed a violet color at the soil surface. These plants were washed in a stream of water and numerous sclerotia were found enmeshed in the superficial hyphae. These could be lifted from the surface of the stem with a needle and showed no connection to the host plant. However, they were deeply entangled in the superficial hyphae from which they arose. The surface hyphae showed a decided tendency to accumulate in strands, proceeding from the infected seed piece upward. While these strands were abundantly connected with transverse hyphae, the fungus showed a marked tendency to progress parallel with the potato stem. This was very striking and quite differ-

ent from that observed upon the original tuber. On the tuber the strands formed a net work which seemed to take no particular direction.

The superficial sclerotia, heretofore mentioned, were rather numerous on the stem. More of them were observed near the ground surface but they were not uncommon on parts deeper down in the soil. There were no evident lesions on the stem of the host. This is very different from a similar attack of *Rhizoctonia solani* which produced long, rather deep lesions, in some cases even extending around the stem, producing a collar rot. When the soil is free from the disease, and infection comes only from the sclerotia on the seed piece, as was the case in this experiment, no conspicuous symptoms of the disease are evident upon the aerial parts of the plant. This may not be true where the soil is thoroughly infected and the plants grown under field conditions.

When the fungus is present in the soil from year to year and can start growing before the potatoes are planted, opportunities for damage are increased. Any serious injury to the under ground part of the plant would undoubtedly be accompanied by above ground symptoms. The tubers, by this time about ½ inch in diameter, began to show the presence of infection cushions (microsclerotia) under the mat of felted mycelium. The root system was very lightly covered with the fungus, but here the hyphae were visible with difficulty to the unaided eye. Occasionally brown specks appeared in the epidermis of the stems and the roots. Microscopic examination showed these to be dead surface cells of meristematic tissue, the injury not extending to the deeper layers of the cortex.

CLIMATIC REQUIREMENTS

Moisture relations. Concerning the moisture requirements of this organism, it is interesting to note the regions from which it has been reported. It has done serious damage in all the countries of western Europe. The disease is reported as developing rapidly under moist conditions in England and France. On the other hand the earliest accounts of the violet root rot disease in America have come from the semi-arid regions of the west. It has been reported outside of this region only within the last decade.

The fact that the disease will develop in the humid areas of Europe, and lately in America, as well as in semi-arid parts of these two continents would indicate that rainfall is not the limiting factor in the spread of the fungus. As a matter of fact, if the organism will thrive under such varied conditions as the reports from this country and Europe show, and attack such an array of hosts as Duggar (6) gives, then it seems that the fungus could adapt itself to many of our important alfalfa, clover, sugar beet, and potato regions.

Temperature. It has been noted by observers both in Europe and North America that this disease appeared in alfalfa fields about the time of the second cutting. In some instances July first has been given as the time the disease first makes its appearance. The plants grown in the preliminary experiment which is summarized in table 2, were grown at temperatures between 90° and 100° F. In fact the greenhouse was being used to force Easter flowers during the first half of the experiment. Five other pots grown at temperatures from 60° to 70° F. did not develop the disease. While this test should not be taken as conclusive, because some other uncontrolled factors may have entered into the result, such as moisture, etc., there is some indication that the fungus develops best at high temperature.

Soil requirements. This disease has been reported on potatoes in Nebraska from regions of sandy soil. This has also been the case, for the most part, where it has appeared on alfalfa in this state. However, it has developed on alfalfa growing in a loam soil. Many writers have made mention of soil acidity in connection with this disease on alfalfa.

INFECTION OF THE HOST

Stem infection of the potato. The superficial surface hyphae produce branches which infect the host tissue by crowding apart the epidermal cells and forcing an entrance. The hyphae enter through the tender epidermal cells of the young dormant buds. While these may not be the only points of initial entrance for the young infecting hyphae, no others were found in hundreds of sections examined. The growth of the mycelium both external and internal, was greatly stimulated in the regions where the hyphae gained entrance to the host. The internal hyphae pursued an intercellular course which was in general, although by no means exclusively, parallel to the surface strands. Internal branching of vegetative hyphae seemed to be no more abundant than the branching of the surface hyphae. These intercellular hyphae permeated the cortex and finally reached the cambium layer. Young hyphal tips initiated the development of internal sclerotia by passing directly through the wall of a host cell and then branching profusely, or by abundant branching in the intercellular spaces, as previously described.

Infection of the young tuber. Early tuber infection follows much the same course as has been described for the entrance of the fungus into the young stem. Many young tubers were examined in all stages of development from slightly enlarged stolon tips to mature potatoes. Here again, entrance of initial infecting hyphae were observed only through the young growing tips such as the stem apex and the "eyes" of the tuber.

The surface cells were forced apart in these regions, and infection took place in a manner exactly as occurred in stem infection. The fungus formed sclerotial cells in the phellogen and these soon broke through to the surface of the tuber. From the exposed surface of this sclerotial cell, hyphae grew out in all directions, joining or fusing with other hyphae from other similarly formed sclerotia or with the superficial hyphae already present on the plant.

Root infection. Root infection of both potatoes and alfalfa was accomplished by the young Rhizoctonia hyphae forcing apart the epidermal cells. The hyphae traversed the cortex and finally reached the cambium layer. In the case of the sugar beet the mycelium soon enters the fleshy succulent root and completely destroys it. The carrot meets a similar fate.

In alfalfa and clover the tap roots are rotted away and the plants soon die leaving more or less circular dead areas in the fields. Upon such crops as alfalfa, where the plants are left in the ground over a period of years, the fungus seems to make more headway and destruction is more complete. Furthermore, if the fungus becomes established in such a field, soil treatment cannot be practiced without killing the plants in the area treated.

This disease no doubt causes some injury to the stems, under field conditions. Observations in the field during the season of 1920 revealed a weakening of the potato plants when the roots were badly rotted. The fungus is evidently at a disadvantage upon a short season crop like potatoes, and the field losses are, as yet, minor in potato fields.

Perhaps the most serious loss to the potato comes while the tubers are in storage. Heavily infected tubers have the periderm broken by the infection cushions and in some cases this breaking is so complete as to split off large portions of the outer skin. This subjects the tubers to drying out. In England, where storage conditions of tubers affected with this disease have been under observation for a number of years, the tubers are reported (1) to become soggy and spongy if kept any considerable length of time. "The mycelial strands originating from the small sclerotia ramify abundantly in the internal tissues, causing a rot which soon reduces the tuber to a pulp."

INOCULATION EXPERIMENTS

Infected tubers covered with the violet felt mycelium were planted in 4-gallon pots in the greenhouse. The soil in eight of the pots was slightly acid in reaction. The soil in the remaining seven pots was well limed. The tubers were planted late in February. Some of the stems were taken for study six weeks later. At that time they were

well covered with the violet mycelium but no sclerotia had formed. Sclerotia were abundant at the end of nine weeks and from these fresh sclerotia isolation experiments were begun with the hope of securing pure cultures of the organism. The results of this work will be reported later.

At the end of the fourteenth week all the potato plants were dug. The infection results are given in table 2. The supplementary crops planted in the pots were alfalfa, sugar beets, radishes, carrots and asparagus. All of these have been reported as susceptible to the violet root rot.

Pots numbered 5–6–7–9–10–11 and 15 were well limed. Liming gave no marked results upon the inhibition of infection. and no noticeable difference in the growth of the host.

The results of the cultural work, as given in the table, show that the disease may be spread by planting the infected tubers. Since the number of "seed" tubers was limited no attempt was made to determine the effect of various seed treatments upon infected tubers when planted in a soil free from this fungus.

Further work must be done before any conclusions can be drawn upon the infection of the supplementary crops here grown.

SUMMARY

1. The violet root rot disease is present in the most important potato growing regions of Nebraska. The disease is of minor importance at present but may become more serious in time.

2. The disease can easily be introduced into new localities by planting infected tubers.

3. Further tests should be made to determine whether the causal organism is identical with that causing a similar disease on alfalfa and sugar beets.

4. Severe infection on the tubers result in a breaking of the periderm which subjects the potato to drying out in storage.

5. This breaking of the periderm is due to the formation of infection cushions below the epidermis which break through to the surface.

6. Cultural experiments to secure pure cultures of the organism are under way.

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SANTO DOMINGO, DOMINICAN REPUBLIC.

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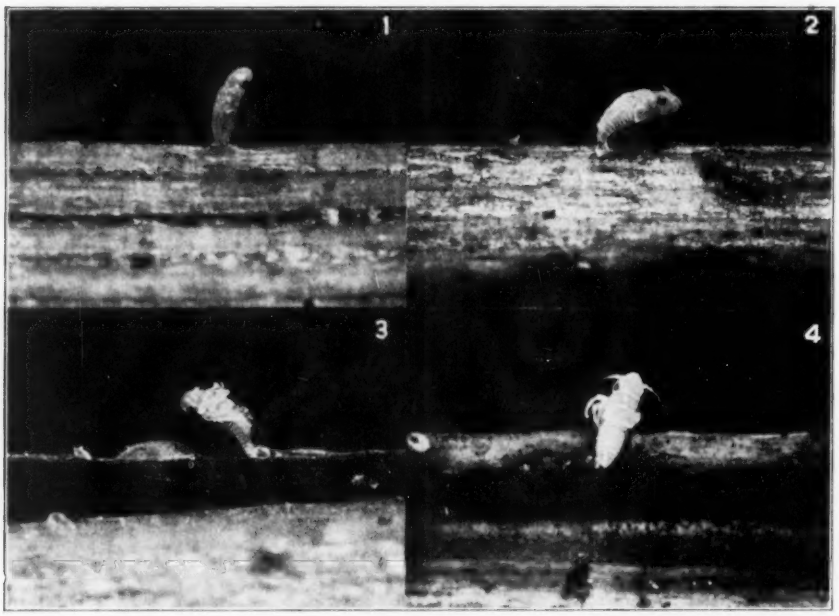
MINIMUM INCUBATION PERIODS OF CAUSATIVE AGENT OF CURLY LEAF IN BEET LEAFHOPPER AND SUGAR BEET

HENRY H. P. SEVERIN

WITH FOUR FIGURES IN THE TEXT

A method of obtaining beet leafhoppers (*Eutettix tenella* Baker) non-infective as to curly leaf (curly top or blight) was first reported in literature by Stahl and Carsner (3). During the process of hatching (Figs. 1–3), the nymphs were transferred with a camel's-hair brush from a curly leaf to a healthy beet and the hoppers failed to produce the disease.



FIGS. 1–4. PROCESS OF HATCHING OF BEET LEAFHOPPER (*Eutettix tenella*)

Fig. 1. Nymph enclosed in chorion, amniotic and vitelline membranes issuing from egg-slit in petiole of beet leaf.

Figs. 2–3. Nymphs after extrication from embryonic envelopes.

Fig. 4. Nymph ready to crawl away. Nymphs in the process of hatching as in figures 1–3, when transferred with a camel's-hair brush from a curly leaf to a healthy beet, failed to produce the disease.

Curly leaf is not transmitted through seeds from "stechlinge" infected with the disease before or after transplanting. The following figures indicate the number of beets grown from such seeds and kept under observations for a period of five months in the greenhouse:

18,154 beets grown from seeds obtained from "stechlinge" blighted before transplanting.
4,232 beets grown from seeds gathered from "stechlinge" infected with curly leaf after transplanting.
352 beets grown from seeds taken from mother beets showing curly leaf symptoms.

22,738

A total of 22,738 beets were grown from blighted seed beets but not a single case of curly leaf developed.

MECHANICAL CARRIER

There is no evidence to show that the beet leaf-hopper is a mechanical carrier of curly leaf. Non-infective nymphs fasted over night, were allowed to feed the next morning on the lower surface of beet leaves showing an advanced stage of curly leaf,—namely the nipple-like papillae and knot-like swellings on the distorted veins. With the use of a hand-lens one could readily see the hopper force its mouth-parts into the leaf as its body swayed from side to side. After each nymph had fed on a diseased leaf from 1–2 minutes, the hopper was disturbed in its meal and transferred to the innermost or youngest leaf of a healthy beet-seedling, where it was allowed to feed for five minutes or less if it completed its meal before the end of that time. This experiment was repeated 44 times; 21 nymphs were used during the first day and 23 nymphs during the second day. Negative results were obtained with 44 beets.

At the end of each day the nymphs were confined in two cages, each enclosing a healthy beet to determine whether they were capable of causing the disease. The 21 nymphs caused a typical case of curly leaf but the 23 nymphs failed to transmit the disease at the end of 40 days. It is evident that the beet leafhopper can become infective by feeding from 1–2 minutes on a blighted beet.

MASS INFECTION

The beet leafhopper is not a mechanical carrier of curly leaf in mass infection of a sugar beet. The method of conducting this experiment was somewhat similar to the preceding except that more nymphs were used and the time of feeding was increased to 5 and 10 minutes on the leaves of curly leaf and healthy beets respectively. Three nymphs were transferred from the foliage of a blighted beet to each leaf of a healthy beet seedling and from 6–21 nymphs were used on one plant. This experiment was repeated with 12 beet seedlings and 131 nymphs, but not a single case of curly leaf developed. A lot of 33 of the 131 nymphs were kept in a cage enclosing a healthy beet to determine whether they became infective. This lot developed a typical case of curly leaf, but five other

batches failed to transmit the disease. It is evident that the beet leafhopper rarely becomes infective by feeding five minutes on a blighted beet.

MINIMUM INCUBATION PERIOD OF CAUSATIVE AGENT OF CURLY LEAF IN BEET LEAFHOPPER

According to Smith and Boncquet (2) the beet leafhopper "does not mechanically transfer the pathogenic factor" when allowed to feed three hours on a curly leaf beet and then transferred for three hours on a healthy beet, or an incubation period of six hours of the disease-causing factor in the insect. The incubation period of the inciting agent of curly leaf in the leafhopper "was no more than 48 and possibly not over 24 hours."

Curly leaf was occasionally produced by an incubation period of 4–6 hours of the causative agent in the beet leafhopper under high temperatures in the greenhouse from April to July. The method used was to confine 25 or 50 non-infective males in cages enclosing curly leaf beets from 1–5 hours, or the adults were previously fasted in empty cages from ½–3 hours, and then allowed to feed from ½–4 hours on blighted beets. These beets showed either the earliest visible symptom of curly leaf,—namely the transparent network of minute veins, or the advanced stage of the disease already described. The cages containing the leafhoppers were then transferred successively to healthy beet seedlings at the end of ½, 1, or 2 hours and in some cases after 24 and 48 hours. In each experiment the insects were finally kept in a cage enclosing a healthy beet seedling for a period of two weeks or longer to determine whether they became infective. Eight typical cases of curly leaf developed of 60 beets used in the following short incubation periods:

Incubation period 4 hrs.	Max. 103° F.	Min. 94° F.	Mean 100° F.
" " 5 "	" 116° F.	" 84° F.	" 102° F.
" " 5 "	" 116° F.	" 84° F.	" 102° F.
" " 5 "	" 120° F.	" 104° F.	" 113.5° F.
" " 5 "	" 120° F.	" 104° F.	" 113.5° F.
" " 5 "	" 120° F.	" 104° F.	" 113.5° F.
" " 5 "	" 122° F.	" 108° F.	" 115.6° F.
* " " 6 "	" 119° F.	" 84° F.	" 104° F.

* In one experiment 25 males with an incubation period of five and six hours caused successive infection in two beets.

Thirty-six batches of 25 or 50 beet leafhoppers with an incubation period of 1–3 hours of the causative agent of curly leaf, failed to transmit the disease to 36 beets. The incubation period from 7–10 hours gave negative results with 29 beets, when the males were allowed to feed one hour on each healthy beet at a mean temperature of 90° F. The incubation period of 24 hours was repeated 11 times and resulted in six curly leaf beets. The experiment with an incubation period of 48 hours was

performed 10 times and seven cases of curly leaf developed. Forty-three lots of 25 or 50 males, confined in cages enclosing healthy beets for a period of two weeks or more, caused curly leaf in 42 beets,—only a single batch of hoppers remained non-infective after feeding one hour on a blighted beet.

Curly leaf was not produced by an incubation period of 1–10 hours of the causative agent in the beet leafhopper under lower temperatures (maximum 80–86° F.) in the greenhouse during October and November. The incubation periods of 17–24, and 48 hours gave negative results with 31 beets at the following temperatures: maximum 80–86° F.; minimum 48–62° F.; mean 67–73° F.

There may be other factors besides temperature, however, which may play an important role on the duration of the incubation period of the inciting agent of curly leaf in the leafhopper In the fog belt districts of Orange County we (1) are inclined to believe that the symptoms of curly leaf do not always appear on the leaves due to a dormant or latent condition of the disease-producing factor in the beet. The usual period of 1–2 weeks for the first visible symptom of curly leaf to appear under high temperatures is often prolonged to over a month or more during the winter in the greenhouse at Berkeley.

MINIMUM INCUBATION PERIOD OF CAUSATIVE AGENT OF CURLY LEAF IN SUGAR BEET

The minimum incubation period of the causative agent of curly leaf in the sugar beet was found to be five days. The method employed was to allow 50 infective males to feed on a healthy beet for one day on March 31. The next day the 50 specimens were removed and 300 non-infective adults were confined in the cage enclosing the infected beet. Twenty leafhoppers were removed daily from the infected beet and transferred to a healthy beet seedling on which they fed for a period of three months. A similar experiment was performed with 50 infective males and 300 non-infective nymphs. In the first experiment the original infected beet showed the earliest visible symptom of curly leaf on April 9, and in the second experiment on April 11. The results are indicated in table 1.

According to table 1, the adults became infective by feeding on the beet five days after it was infected and the nymphs at the end of eight days. A glance at table 1, shows that 20 adults which were transferred to a healthy beet on April 7, after feeding seven days on the infected beet, failed to transmit curly leaf. It is evident that non-infective hoppers became infected by feeding on a sugar beet infected with curly leaf before the cleared veinlets on the youngest leaf appeared on April 9, 11.

Experiments similar to the preceding were repeated 10 times and in one other case the incubation period of the causative agent of curly leaf

in the sugar beet was five days at the following temperatures: maximum 108° F.; minimum 51° F.; mean 71.7° F. The infected beet showed the first visible symptom of curly leaf at the end of 10 days.

In the next experiment instead of the nymphs and adults feeding separately on two infected beets as in the first experiment, both were confined in a cage enclosing a single beet, infected one day previously by 50

TABLE 1

Incubation periods of causative agent of curly leaf in sugar beets

DATES, 20 NYMPHS AND 20 ADULTS WERE TRANSFERRED TO HEALTHY BEETS	DATES CURLY LEAF DEVELOPED (ADULTS)	DATES CURLY LEAF DEVELOPED (NYMPHS)	TEMPERATURE		
			MAX. °F.	MIN. °F.	MEAN °F.
(Mar. 31)			86	56	71
Apr. 1			96	58	77
Apr. 2			80	50	65
Apr. 3			104	56	80
Apr. 4			108	50	79
Apr. 5	May 4		88	50	65
			93.6	53.3	72.8
Apr. 6	Apr. 22		86	52	69
Apr. 7			86	50	68
Apr. 8	May 29	June 4	100	48	74
			90.6	50	70.3
Apr. 9	Apr. 26	Apr. 24	92	52	72
Apr. 10	May 3	Apr. 20	86	54	70
Apr. 11		Apr. 27	95	51	73

infective males. Twenty adults and 20 nymphs were removed daily from the infected beet and transferred to cages enclosing two healthy beet seedlings. The original infected beet showed the first visible symptom of curly leaf nine days later, but the adults became infective at the end of seven days (maximum 119° F., minimum 46° F.; mean 71° F. temperatures) and the nymphs at the end of nine days (maximum 119° F., minimum 46° F., mean 76.4° F. temperatures) coincident with the appearance of curly leaf in the infected beet.

Before molting insects stop feeding and empty the contents of the alimentary canal. During the process of molting the chitinous intima of the fore and hind-intestines as well as the ducts of the salivary glands are shed. The nymphs, however, do not lose their infectivity during the process of molting, so this would hardly explain the longer period by the nymphs to become infective.

Mr. C. C. Epling in his thesis work found that the incubation period of the disease-producing agent of curly leaf in "stechlinge" and beets about three months old was prolonged during the winter. The nymphs became infective at the end of 15 days (maximum 102° F.; minimum 58° F.; mean 72° F. temperatures) by feeding on a "stechling" infected by 50 infective adults. The infected "stechling" developed curly leaf at the end of 16 days. In the case of beets about three months old, the nymphs became infective at the end of 15 days (maximum 94° F.; minimum 58° F.; mean 71.7° F. temperatures) and the infected beet showed the transparent venation of curly leaf at the end of 14 days.

During the past three years the work on the beet leafhopper and curly leaf was conducted in cooperation with the departments of Entomology and Plant Pathology of the University of California.

SUMMARY

The beet leafhopper when it hatches from the egg is non-infective.

Curly leaf is not transmitted through the seeds from "stechlinge" affected with the disease before and after transplanting.

The beet leafhopper is not a mechanical carrier of curly leaf, nor a mechanical carrier in mass infection of a beet.

The minimum incubation period of the causative agent of curly leaf in the beet leafhopper required four hours at the following temperatures: maximum 103° F.; minimum 94° F. and mean 100° F. and five days in the sugar beet at the following temperatures: maximum 93.6° F.; minimum 53.3° F. and mean 72.8° F.

CALIFORNIA AGR. EXP. STATION.

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BRIEFER ARTICLES

THE FALSE MILDEW OF TOBACCO INTRODUCED INTO THE UNITED STATES FROM THE DUTCH EAST INDIES?

B. T. Palm

In a recent publication Smith and McKenney[1] have described in some detail the occurrence in the United States of America for the first time on tobacco of a dangerous tobacco parasite, *Peronospora hyoscyami* DeBary. As the disease is causing considerable anxiety in the infected tobacco districts the authors discuss the possible means by which the introduction of the fungus in question has been brought about.

After stating that *Peronospora hyoscyami* has been reported to attack *Hyoscyamus niger* in Europe, tobacco in Australia and South Africa and *Nicotiana glauca* in California and Texas, they proceed (p. 4):

"No definite statement as to the probable means of introducing the disease into the United States can be made at present. However, it must be borne in mind that the mats used in baling tobacco in this region are imported annually from Sumatra, arriving in the month of September, about the time the harvested seed is being cleaned and stored away. It is known that certain insects have been introduced into the United States on such mats and it is not impossible that this disease may have been introduced into the United States through this agency, especially as many of the mats are second hand, having been previously used on East-Indian tobacco." "On the other hand," they suggest, "the disease may have come into Florida by the way of California and Texas or from the south Atlantic or Gulf seaboard where *Nicotiana glauca* also grows."

The importance of the disease in question makes it necessary to elucidate the sources of importation and therefore it may not be superfluous to discuss once more the chances for an importation of the fungus, by means of the so-called Sumatra tobacco mats.

The mats used in packing tobacco are manufactured from one or more species of sedge grass either cultivated or collected in the wild by the natives in the low swampy plains of South Borneo. As far as the writer has been able to ascertain during a visit to that part of Borneo no tobacco is cultivated there. The new mats are then shipped from Bandjermasin, the harbour town of South Borneo, to the United States of America directly or via Holland. It does not seem very likely then that in this way a parasite of the Peronospora type could be introduced.

The second hand mats have another history.

[1] Smith, Erw. F. and R. E. B. McKenney. A dangerous tobacco disease appears in the United States. U. S. Dept. of Agriculture, Circular 174. April, 1921.

Shipped from Bandjermasin to Java or Sumatra, they are stored on the tobacco estates there. After baling of the tobacco on the estates every package as a rule is disinfected with carbon dioxide (150 ccm³ p. M³ for 48 hours) against Lasioderma. The bales then are shipped to Holland where they are stored in the tobacco warehouses awaiting the auction sales. From Holland the tobacco packages go to the United States, where the mats are sold to the tobacco growers.

An introduction of the tobacco Peronospora through the intermediary of the second hand mats would, theoretically, be possible in three ways: from the Dutch East Indies (Java and Sumatra), by contamination during shipping, and from Holland.

The first possibility can from our present state of knowledge not be absolutely discarded; the chances seem, however, very small as *Peronospora Hyoscyami* as yet has not been found on tobacco and, as far as I know, on no other plant in the Dutch East Indies.

Thus far, no genuine species of Peronospora has, as far as the knowledge of the writer goes, ever been found in the Dutch East Indies. In view of this, it is true, negative evidence Java and Sumatra will probably not prove to be the place of origin of the tobacco "blue mold" found in the United States. The tobacco diseases in Java and Sumatra have been rather extensively studied during the last twenty years and it's not very probable that a Peronospora of tobacco would have escaped notice for so long a time. The writer himself has during more than five years, as pathologist, repeatedly visited the various tobacco districts in Java and during the last year made a special survey of the Sumatra tobacco diseases, without having been able to find the tobacco Peronospora, either on tobacco or wild Solanaceae.

The possibility of contamination of the packing mats during shipment by tobacco from other countries might not, at present, be wholly disregarded. However, as a rule every possible precaution is taken to exclude other cargo from the rooms where Sumatra and Java tobacco has been stowed, this principally to avoid contamination *en route* by Lasioderma. There is every reason to think, therefore, that tobacco from other tropical countries, as British North Borneo, China and British India—which perchance may be shipped on the same ships as Dutch East Indian tobacco—also will be kept separated from the latter.

There remains now to be considered the third stage during the transportation of the mats where a contamination might take place, that is in Holland. During storage in the warehouses there, tobacco from other countries as Brazil, Asia Minor and Europe might occasionally come into contact with the Dutch East India tobacco. It is of course very difficult to estimate the chances for an infection. Taking into consideration,

however, that such a long distance spread of Peronospora in this case as well as during shipping must be solely based on a contamination by resting spores which may adhere to the bales, the chances would seem exceedingly small for an infection to be brought about in this manner.

The evidence brought forward above is decidedly not in favour of the theory of an introduction of *Peronospora Hyoscyami* De Bary into the United States from the Dutch East Indies by means of second hand tobacco mats even if it can not at present be absolutely disproved. The second theory advanced by Smith and McKenney, namely that *Peronospora Hyoscyami* has been introduced into Florida either from California and Texas from where this fungus already has been reported or from the South Atlantic or Gulf seaboard where *Nicotiana glauca* grows, would, however, appear to be the more plausible one.

DELI EXPERIMENT STATION
MEDAN, SUMATRA, DUTCH EAST INDIES.

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